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A TRIBUTE TO DR. ROBERT B. OSGOOD

INTRODUCTION

BY FRANK R. OBER, M.D.

IN 1931, Dr. Robert B. Osgood resigned from the John Ball and Buckminster Brown Professorship of Orthopedic Surgery at the Harvard Medical School and, at the same time, he also resigned as Chief of the Orthopedic Service of the Children's Hospital, Boston, after a service of nearly nine years.

Very soon after this, the members of the Orthopedic Staff, who had served under Dr. Osgood, agreed to produce an article on some phase of work in which they had been interested during their period of service with him. These articles were to be published in a complimentary volume to Dr. Osgood as a token of appreciation of his many kindnesses and for his stimulation to all of us who were privileged to be his associates.

It seemed especially fitting that we ask Sir Robert Jones, a close friend and admirer of Dr. Osgood, to write the foreword of this volume. Needless to say, Sir Robert agreed to do so with alacrity and pleasure.

The articles in this volume were produced, for the most part, from material obtained at the Children's Hospital, Boston.

FOREWORD

BY SIR ROBERT JONES*, K.B.E., C.B., F.R.C.S., F.A.C.S.

I FEEL it a great honour that I am considered worthy to write a foreword to this volume presented to R. B. Osgood by his friends. There must be so many who could more worthily fulfill the trust but very few who have so deep an affection for him, or a greater admiration for his qualities of heart and mind. I have a vivid recollection of him as a young man when he used to visit me in Liverpool; keen and enthusiastic, full of courteous enquiry he impressed me at once as exceptional. He was then a wholehearted searcher for truth, and to this day truth has remained his sole objective. Even in those days—at an age when youth is so sure of most things—he always remained modest and displayed a strictly introspective mind, both thoughtful and provocative. During all the years that have passed since those early days he has exercised the same charm over all who have come in contact with him. With marked independence and self-reliance he does not possess any element of aggressiveness in his varied armamentarium, and yet I know of no surgeon who can reach his objective so surely and so often. He has left no trace of bitterness in his path to fame and no one can recall an unkind word or thought, for he has always been the most understanding and kindly of writers. He is a gentle critic, of calm and sound judgment and not a controversialist in the ordinary meaning of the term.

His logical mind—tempered and enhanced by a delightful imagination—has overcome all obstacles and he has never given cause for resentment. He has always been singularly unfettered by tradition and can throw light upon obscure problems from many unexpected angles. I remember when an animated discussion took place on fractures at a large meeting in England he contributed a graceful and persuasive speech which Lord Moynihan—himself a great orator—described to me as one of the most delightful and convincing speeches to which he had ever listened.

There is no one in America who has so captivated the British Orthopaedic Surgeons as Robert Osgood. This is due not only to his personality, but also to the two great services he has rendered to this country. The first was the founding of the British Orthopaedic Association, and the second his great sacrifice of health and energy on behalf of our soldiers during the war. They are both unforgettable acts and we owe him a deep debt of gratitude which cannot easily be repaid. He graciously consented to be my second-in-command during and after the establishment of Orthopaedic Centres, which ul-

*Deceased January 14, 1933.

Jones—See "This Week's Issue," page 105.

timately housed over twenty thousand wounded men. His work during this period was colossal. He not only helped in organising but he visited centres all over the country inspecting the work, training the men, settling disputes, changing chaos into harmony. All this was effected with such tact and understanding that a welcome was accorded him everywhere. He was with me when Goldthwait arrived with the wonderful gift of 26 young and skilful orthopaedic surgeons—the gift of America for the duration of the war. It is impossible to overstate what that gift meant to us. It saved a desperate situation, and the supervision of Goldthwait and Osgood over that contingent set a high standard upon the work they did. The foundation of the British Orthopaedic Association was at the direct instigation of Osgood who helped us to draw up the rules and attended the first meeting of the Society under the presidency of Muirhead Little.

It is no wonder, therefore, that we have a great admiration for Osgood and that he has so warm a corner in the hearts of our people, for it is he, more than any other, who has helped to forge a close link between American and British Orthopaedic Surgeons. During the war he was an Ambassador of Peace and Goodwill to his British colleagues.

I need not dwell upon his contributions to surgery for they are known to all of us. They deal with many branches of the art he loves so well, and are excellent examples of lucid expression couched in graceful English. Neither is it necessary to refer to the many important academic positions he has filled, his hospital services, nor the special lectures he has delivered in this and other countries. They have made a distinct impress upon the art of surgery and enhanced its literature.

I could linger upon his influence over students, practitioners and colleagues were it not so generally known. He is mentioned in terms of enthusiasm and affection by all who have visited me in England, but it is what we expect who know his high ideals and scholarly mind. He radiates an atmosphere of friendliness, encouragement and hope, and is inspired with a loyalty to all that is ennobling. His life is a stirring example to all of us who need moral stimulus; it is indeed a joy to be admitted to his circle of intimate friends.

His character is full of facets, and one of the most outstanding is his love of simplicity. Those of us privileged to have spent a holiday with him and his charming and devoted wife and child in their country cottage will know what I mean. There family life is an idyll. Fresh air, simple tastes, devotion to animals, surprise visits to neighbours with similar tastes make up their days. The sense of humour and of fun, the boyish pranks, and above all this a true love of the best literature make a lasting impression upon my mind.

Although his hospital service has come to an end his activities must persist through life. May he long be found amongst us to continue his countless services and to remain an inspiration to his admirers the world over.

TENDON TRANSPLANTATION IN THE LOWER EXTREMITY

BY FRANK R. OBER, M.D.*

GENERAL CONSIDERATIONS

TENDON transplantation has lost a great deal of the position it formerly held, since bone stabilizing operations have advanced so much, especially during the past ten years. In spite of all the modern bone work which is being done in so many of the orthopedic clinics of the world, tendon transplantation must have its place, not only as an adjunct to well-planned architectural reconstruction (for which we owe Michael Hoke a great deal), but also in many instances where operations on bones are impossible or unwise. Surgeons have become so profoundly interested in the excellent results of arthrodeses that only a very few have undertaken to improve and develop a better technique of tendon transplantation. It therefore has come into disrepute in many clinics, chiefly because the technique of bone fusion has developed to such a high degree. The few surgeons who have stuck to tendon work and used arthrodesing operations as an adjunct are, from their results, more than justified in not eliminating the valuable aid a transplanted tendon may give an improving function.

We owe a great deal to those pioneers who first did tendon work—Nicalodini, and later Langé, and still later Biesalski; and Leo Mayer, who has done so much original work on the anatomy and physiology of tendons, which has made a distinct advance in the technique of tendon transplantations. The writer has attempted, during the past thirteen years, to simplify the technique of the above workers, and, in so doing, tendon transplants have been done which were formerly thought to be impossible, e.g., transferring the posterior tibial forward to act as a dorsal flexor.

With few exceptions, tendon transplants about the ankle joint must be combined with an arthrodesis for two reasons. First, because removing a tendon from the lateral aspect of the ankle joint and conveying it to another position in-

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creases the instability of the joint and, secondly, because stabilizing the joint permits the tendon to divorce itself from its former function of stabilization and speeds up its new function.

The requirements necessary for a successful physiological outcome may be summarized as follows:

1. Selection of suitable cases.
2. Careful study of the physiology and anatomy of each case.
3. Correction of existing deformity before proceeding with the transplant.
4. Selection of a suitable tendon or tendons.
5. Prevention of adhesions.
6. Leverage.
7. Method of insertion of tendon.
8. Direction of tendon and muscle.
9. Tension.
10. Wound closure.
11. Diligent and careful postoperative treatment.

1. *Selection of a suitable case.* There must be sufficient power in all the tendons present so that when one or more of these are taken there will be enough power left in the others for them to function properly. In some cases, when stabilization of the joint has been done, a partially paralyzed tendon left in its original position may exert enough pull to produce a deformity. When such a fact is obvious, the partially paralyzed tendon should be placed in a position to improve function.

2. *Physiology and anatomy.* The general gait and position of the foot must be studied very carefully in order to determine what the requirements are for obtaining the best physiological balance in a paralyzed foot. The kind and degree of deformity must be noted and whether the deformity is fixed or due to relaxed ligaments and unequal muscle pull. The amount of power in each group of muscles must be estimated as well as possible.

3. *Correction of deformity.* If the deformity is fixed, it must be corrected at a previous operation or at the same operation, provided it is done before the tendon is implanted. A short tendo Achillis may often be corrected manually; rarely does it require open lengthening. A contracted plantar fascia may be tenotomized. Fixed ligamentous deformities may be reduced manually or by means of a Thomas wrench. Fixed bony deformities can be corrected by osteotomies, arthrodesis, and astragalectomies.

4. *Selection of a suitable tendon or tendons.* Where it is possible, one should select a tendon which has nearly as much power as the one it is to replace. In case this is impossible there should be enough power to balance its opponent.

5. *Prevention of adhesions.* One of the greatest bugbears in tendon transplantation is the formation of adhesions. These may be avoided or minimized by careful handling of the tendon, carrying the tendon through the compartment of the paralyzed tendon, and meticulous wound closure.

6. *Leverage.* Te secure normal leverage one must always transplant at or opposite the insertion of the paralyzed tendon whenever practical. For example, transplanting the peroneus longus to the scaphoid to relieve the paralyzed anterior tibial is doomed to failure because of loss of normal leverage and because that is not the normal insertion of the anterior tibial. If there is not sufficient power in the transplant to function, when placed at or near the insertion of the paralyzed tendon, then it must be cut long enough so that it may be transplanted at a point where the increase in leverage will make up for loss in power.

7. *Method of insertion of tendon.* The transplant may be secured in one of three ways: tendon to tendon, tendon to periosteum, or tendon to bone. In the first, the tendon is apt to slide up proximally, and in the second, the periosteum often stretches, allowing the transplant to become elongated and so lose contractile power. The third is ideal, as the transplant practically always becomes firmly united to the bone. Heavy white silk is a satisfactory suture for uniting the tendon to the bone, since it retains full tensile strength for an indefinite period. The distal end of the transplant is well scarified.

8. *Direction of tendon and muscle.* In transplanting a tendon, plan to have it follow as nearly as possible the course of the paralyzed tendon, avoiding right angle and short oblique directions, as one must assume that angulations interfere with muscle cell function and also interfere with the physics of pull. If the muscle and tendon follow the anatomical course of the paralyzed muscle, then the physiological result ought to be better and brought about more quickly.

9. *Tension.* There is no practical way of measuring this, but, as a general rule, the transplant should be under enough tension to hold the foot in a corrected position. It has been pointed out by many workers that too much tension results in overstretching, and overstretching causes fatigue with resulting loss of power. If the tendon is not under any tension, then the slack will not be taken up by the muscle belly, and again there is not enough power to function.

10. *Wound closure.* In closing wounds fine black silk has been found to be a satisfactory suture material, first, because there is a minimum of inflammatory reaction, and, secondly, because one can secure a very neat approximation of all tissues with a minimum amount of material. It is very necessary to unite gliding tissues over the transplant in order to avoid the formation of adhesions.

11. *Postoperative treatment.* In transplants

of the extensor proprius hallucis to the neck of the first metatarsal, and in transplants of the posterior tibial, flexor communis digitorum and peroneus longus to the os calcis, it is not necessary to apply retentive measures, and exercises to reëducate these muscles may be started forty-eight hours after the operation. In other transplants exercises may be started in ten or twelve days, leaving apparatus on the foot until the transplant shows good function. When stabilizing bone operations have been combined with transplanting, the exercises should not be done until bony union is solid, i.e., six to eight weeks after the operation.

TRANSPLANTATIONS ABOUT THE ANKLE JOINT

Indications. Tendon transplanting may be done for paralysis of the anterior tibial, anterior and posterior tibials, peroneals, and gastrocnemius. Where all the muscles have normal power except the anterior tibial, the extensor proprius hallucis may be used if there is fair function in the anterior tibial but not enough power to hold the foot in the normal walking position. The extensor proprius hallucis may be implanted into the neck of the first metatarsal bone, passing the cut end of the tendon through a drill hole made from above downward. The tendon entering the hole at the inferior medial aspect is withdrawn and the end sutured to the capsule and bone at its exit. The tendon should not be sutured on itself as the power of inversion and adduction of the foot is lessened in so doing. The distal portion of the tendon is sutured to the extensor brevis digitorum to avoid a hammer toe deformity. This operation may be done with a local anesthetic.

When the anterior tibial is totally paralyzed, the peroneus longus may be transplanted as follows:

a. The junction of the peroneus longus muscle and tendon is exposed on the lateral aspect of the leg. (Fig. 1.)

FIG. 1. Peroneal longus tendon transplant. Lower wound shows tendon exposed. It is divided under the foot. Upper wound shows muscle and tendon being separated from the peroneus brevis muscle.

b. The lower end of the tendon is exposed on the lateral aspect of the foot. (Fig. 2.)

FIG. 2. Tendon being withdrawn. The muscle should be grasped with a wet sponge.

c. The base of the first metatarsal and internal cuneiform bones are exposed on the medial aspect of the foot and an osteoperiosteal bone bridge is raised with a chisel in the base of the first metatarsal near the insertion of the anterior tibial. (Fig. 3.)

FIG. 3. Groove being made through base of first metatarsal.

d. The peroneus longus is divided distally, well under the sole of the foot, so that about one-half an inch of the dense fibrous portion is left at the end of the freed tendon.

e. The tendon and muscle are freed from the peroneus brevis muscle, the tendon sheath, and the fibula. (Fig. 4.) An X suture of number 16 silk is placed in the dense fibrous end of the tendon.

FIG. 4. Tendon being passed down under the deep fascia through the anterior muscle compartment.

f. The septum between the peroneus longus muscle and the anterior tibial compartment is broken through, and, by means of a carrier,

passed down through the anterior tibial compartment the tendon is drawn out of the wound at the medial aspect of the foot. (Fig. 5.) The

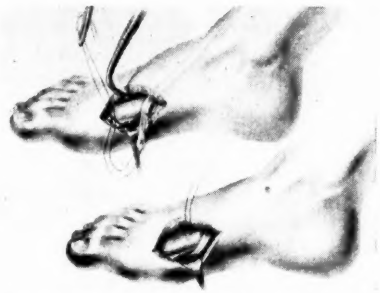

FIG. 5. Upper drawing shows stitch in dense bulbous portion of the tendon.
Lower drawing shows tendon inserted into bony groove.

silk is drawn through the hole in the bone from below upward (Fig. 6), the end of the tendon just emerging at the upper end of the opening

FIG. 6. Method of suturing to the bone.

and firmly sutured to the bone and ligaments in the neighborhood and not on itself, thus simulating as well as possible the insertion of the anterior tibial tendon.

D. S. No. 11329.

FIG. 7. Peroneus longus tendon transplanted to base of first metatarsal. Eight years after operation.

If the posterior tibial tendon is also paralyzed, the extensor proprius hallucis must also be transplanted as first described.

In paralysis of the peroneals, the anterior tibial tendon with some attached bone is passed through the sheath of the common extensor and sutured to the base of the third metatarsal bone, first having raised an osteoperiosteal flap from the proximal end of the bone so that the cut end of the tendon with its bone flaps is in contact with new bone.

In paralysis of the gastrocnemius the peroneus longus and posterior tibial tendons are freed as described for peroneal transplants, and a transverse hole is drilled through the tip of the os calcis near the insertion of the tendo Achillis. These tendons are passed down through the tendo Achillis compartment, *crossing behind the tendon,* and passed through a drill hole in the os calcis, the posterior tibial from without inward, and the peroneal from within out-

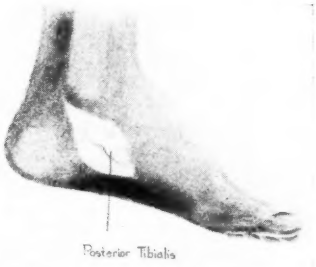

FIG. 8. Exposure of Posterior Tibial Tendon. A piece of the scaphoid bone is removed with the tendon.

ward. The cut ends are sutured to the os calcis and the tendo Achillis. If the flexor communis digitorum is not paralyzed, it should also be used as it adds a great deal to the success of the operation, as Naughton Dunn has shown. The function of plantar flexion will be increased a great deal if the Hoke type of arthrodesis is

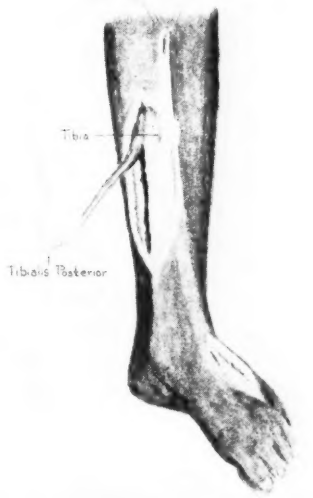

FIG. 9. Shows belly of posterior tibial muscle dissected free from the tibia. The belly is passed into the anterior muscle compartment.

done, being careful to displace the foot backward in order to obtain more leverage. (See photos.)

The anterior tibial may be used as a posterior transplant if there is weakness in the calf group, including the posterior tibial and the peroneals. This transplant may be done if there is good power in the long extensors of the toes and,

when combined with a Hoke arthrodesis, results in fair function and a well-balanced foot. This operation was first done by Ralph Ghormley, who was formerly associated with me.

The tensor fasciae latae may be substituted for the gastrocnemius. The iliotibial band is exposed through a lateral incision extending upward from the head of the fibula for a distance of five to six inches. The iliotibial band is dissected upward in a strip one inch wide, from the surrounding structures. A second incision three inches long is made over the junction of the tensor and the fascia lata. The two openings are connected subcutaneously. The distal portion of the flap is drawn upward and out

FIG. 10. Posterior tibial tendon inserted into base of third metatarsal bone.

of the upper wound, and the muscle belly of the tensor is separated from the surrounding structures. Beginning at the muscle junction of this flap, a piece is split off the fascia from above downward, and if this is not long enough to reach the os calcis, a second strip is dissected from below upward in a similar manner, the whole structure resembling a threefold rule being elongated. Two number 16 silk sutures are placed at the end of each fold so they will not tear apart. With a tendon carrier this elongated tendon is drawn from the thigh and out the lower wound. A third incision two inches long is made over the lateral aspect of the tendo Achillis and the os calcis. The carrier is next inserted into the second wound and passed beneath the deep fascia of the leg, emerging at the third wound. A flap of bone is lifted off the os calcis near the insertion of the tendo Achillis, and while an assistant holds the thigh and leg flexed at 90°, the end of the tendon is inserted under the bone flap and firmly sutured there and also to the tendo Achillis.

There has been a good deal of adverse discussion about transplanting the posterior tibial tendon forward. In cases of equino varus where there is normal power in the gastrocnemius and the posterior tibial muscles, the latter is the chief deforming factor. Tenotomy of the posterior tibial and correction of the varus deformity relieve the situation in part, but it is difficult to prevent the recurrence of equinus and it seems an unwarranted waste to throw away the power of a good muscle.

It has been found that the posterior tibial can be transplanted forward with success in the

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FIG. 11. Posterior tibial transplant to base of third metatarsal. Hoke arthrodesis of ankle. One year after operation.

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FIG. 12. Posterior tibial and peroneus longus tendons transplanted backward to tip of os calcis. Hoke stabilization. One year after operation. Walking on toes.

following manner. An incision is made over the medial aspect of the ankle joint exposing the attachment of the posterior tibial muscle to the navicular. The tendon is freed from the bone, including a thin bone flap. A second incision is made over the junction of the posterior tibial tendon and muscle junction. The tendon is withdrawn from the upper wound and the muscle belly is dissected well up the tibia. The periosteum on the medial surface of the tibia is incised in an oblique direction and pushed off the bone so that the belly of the posterior tibial muscle will be in contact with bare bone, and the anterior tibial compartment entered. A third incision is made over the base of the third metatarsal. The posterior tibial tendon is now drawn down through the anterior tibial compartment in such a way that the muscle belly of the posterior tibial is in contact with the bone surface of the tibia, being careful that

the muscle and not the tendon is in contact with the bone. The distal end of the tendon is implanted into a groove in the base of the third metatarsal. This operation should be combined with a Hoke arthrodesis to secure lateral stability and prevent recurrence of the deformity.

TRANSPLANTATION ABOUT THE KNEE JOINT

The quadriceps muscle is more frequently paralyzed in the thigh than any other. The two muscles most often spared are the sartorius and the tensor fasciae latae, and their use as knee extensors is fairly satisfactory and causes less disability than transplanting the ham-

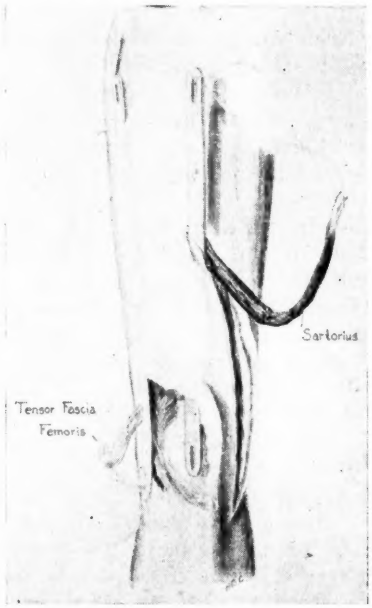

FIG. 13. Shows iliotibial band drawn under fascia lata and sartorius ready to be drawn through.

strings. There is a danger of the knee hyperextending when one or more of the hamstring muscles are used. The tensor through its gluteal attachment helps to extend the knee and the anatomical situation of this muscle and the sartorius gives a better physiological action on the patella and is in better anatomical relation to the anterior structures of the thigh than is a hamstring.

Technique of the operation. A curved incision eight inches long beginning over the ligamentum patella is made, curving outward and upward along the iliotibial band. The iliotibial band is freed from the fibula and from its surrounding structure as a flap three-fourths of an inch wide. A second incision three inches long is next made over the lower part of the tensor. The two wounds are connected by a subcutaneous tunnel, and the loose flap is drawn out of the upper wound. The muscle belly of the tensor is now freed from the surrounding structures. The skin is next reflected medially from the patella and its tendon, exposing the fascia over the lower end of the quadriceps tendon. This fascia and the aponeurosis over the patella are split longitudinally, cutting part way into the tendon of the quadriceps and into the patella substance and extending down into the patella tendon. These two cut edges are reflected to each side for half an inch. A third incision five inches long is made over the medial aspect of the thigh from above downward, ending at the adductor tubercle. The knee is flexed to a right angle, the belly of the sartorius is exposed and freed from its surrounding structures, and the tendon is severed at its insertion. An X suture of silk is placed in the end of this tendon and another in the end of the iliotibial band. A carrier is passed beneath the fascia lata, emerging at the opening just over the quadriceps. The silk is inserted into the eye of the carrier and the new tendon of the tensor is drawn out of the lower wound. The muscular septum between the sartorius and the quadriceps is next perforated, and, by means of a tendon carrier, the sartorius is also drawn out of the wound over the quadriceps tendon. While an assistant holds the leg extended and the thigh flexed 45°, the sartorius tendon is

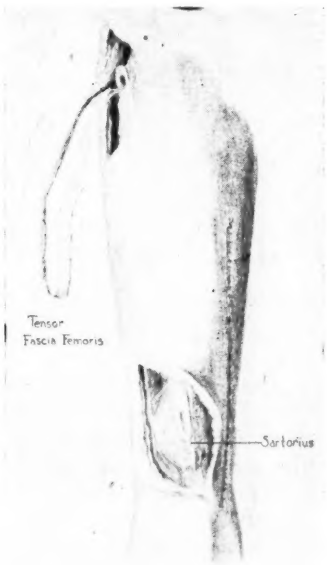

FIG. 14. Shows sartorius drawn through and implanted. The iliotibial band is being drawn to its new insertion.

firmly sutured to the patellar tendon. The iliotibial band is next pulled down over the exposed sartorius, and it, also, is firmly sutured to the patellar tendon with number 16 silk. Each edge of this band is sewed to the reflected edges of the aponeurosis of the patella and to the edge of the sartorius muscle which lies on the bare patella. When the operation is finished the leg will remain extended on the knee, if the thigh is supported just above the joint. Note that the transplants are placed under the fascia lata and not subcutaneously, as is so often done.

In cases of weakness of the hamstrings, the tensor fasciae latae may be displaced backward along the course of the biceps femoris and attached medially to the biceps in the head of the fibula. This operation can be combined with a tenodesis of the biceps to the femur in cases of genu recurvatum.

TRANSPLANTATIONS AT THE HIP

There are two hip conditions which are very disabling—paralysis of the extensors and paralysis of the abductors. These may occur singly or in combination; when they occur together, the hip joint may dislocate.

FIG. 15. Shows sartorius and iliotibial bands sutured to ligamentum patella. Reflected aponeurosis and periosteal flaps of patella being sutured to the transplanted tendons.

In a pure abductor paralysis, the technique of Legg gives excellent results. Formerly Legg advocated inserting that part of the fascia lata just below the tensor into the lateral aspect of the femur about two or three inches below the tip of the trochanter. In many cases the results of this procedure were excellent; but, as the insertion of the tensor is a little forward of the center of gravity, the full abduction power of the tensor was not always obtained, so lately Legg has advocated transplanting the origin of the tensor backward in order that the line of pull may be in line with the long axis of the femur. This operation gives better results than the former operation; but the surgeon has to be very careful to avoid injuring the superior gluteal nerve, and the suturing of the upper portion of the tensor to its new position is a little difficult, since the line of suture is below the crest of the ilium and into fascia. The writer has modified Legg's technique in several instances with good results. The anterior border of the tensor is mobilized and the origin is divided along the crest of the ilium to a place just above the trochanter. The belly of the muscle is next freed from the deep structures and the whole muscle is turned backward on itself so that the anterior edge is sutured to the fascia behind the trochanter. In this way there will be a straight pull from the crest to the insertion of the iliotibial band. The upper edge is sutured to the underlying intact fascia.

Paralysis of the gluteus maximus gives a very disabling and unsightly limp and it also is very fatiguing to the patient. Seven years ago the writer described an operation to help relieve this paralysis. A large number of patients have submitted to this operation, many of them with double hip flexion contractures and with no power below the waist. All of them have been helped to a marked degree, but only a few have had complete relief from their disability. Several of these patients can get about the house without crutches. All of them can get out of chairs more easily. They can navigate stairs a great deal better, and the tremendous backward body thrust in walking has been relieved to a great extent in the majority of instances. In other words, there has been a definite improvement in function, which is very important in any crippling condition.

In the early operation, the iliotibial band was mobilized beginning at its attachment on the lateral aspect of the fibula, and extending upward to the muscle belly of the tensor fascia lata. This muscle was freed at its anterior and posterior aspects, and the iliotibial band was drawn through a drill hole made through the shaft of the femur opposite the lesser trochanter. The free end was then passed up over the gluteus maximus and sutured to the outer three-fourths of the erector spinae group. In a later development of this operation the free flap of fascia is passed under the muscle and periosteum two inches below the base of the trochanter, firmly sutured to this at the entrance anteriorly, and to the gluteus maximus tendon posteriorly. The new tendon is passed upward over the gluteus maximus and sutured to the erector spinae group, which has been previously mobilized in the following manner: the lower five or six inches of the erector spinae are exposed. The erector spinae muscle is split longitudinally in a line of cleavage about one-half inch lateral to the spinous processes of the last three or four lumbar vertebrae, extending down over the sacrum so as to free the tendinous portion of this muscle from the sacrum and posterior crest of the ilium. The whole muscle is separated along the lateral intermuscular septum and extending its whole depth down to the transverse processes and upward as far as the perforating dorsal nerves. The iliotibial band is secured to the under side of this muscular flap on either side and overlapping for about two to three inches. Many of these patients have had a severe lumbar lordosis, which in one case

had persisted for seventeen years. In all cases this severe lumbar lordosis entirely disappeared.

SUMMARY

1. Tendon transplantations at the ankle improve function if the cases are well selected and the operative technique is carefully followed out.
2. Tendon transplants combined with stabilizing operations are more useful because better stability results in better muscular control.
3. Many transplants may be done now which were formerly thought to be valueless.
4. Tendon transplants at the knee result in improved function and better security and stability of the knee.
5. The sartorius and tensor fasciae are the more ideal muscles to use; first, because of their anatomical situation, and secondly, the function of these two muscles is not so opposite as that of the hamstrings.
6. Transplants at the hip result in improved function and minimize an unsightly and troublesome gait.

A NEW OPERATION FOR SLIPPING PATELLA

BY ROBERT SOUTTER,* M.D., F.A.C.S.

SUCCESS of this operation in the long run depends on the technique and the mode of attachment which must stand all kinds of strains with the knee in any position. The points of attachment should be chosen to give the greatest mechanical advantage. The method here described has been used on a large number of adults and children with success.

This method was first used by the author in congenital dislocations of the patella at the Children's Hospital, Boston. Since then, both adults and children have been operated on by the same method. It is so much simpler than changing the angle of the condyle by cutting the femur and setting it at an angle, cutting away half of the patella ligament and transplanting half, reefing the capsule which is useless, and many elaborate methods which involve alteration in the existing tissues or opening the joint.

Gallie has well demonstrated the use of fascia as ligaments and for the repair of torn or overstretched tissues. As a procedure, it is now well established. Its specific use in dislocation of the shoulder and dislocation of the patella has now proved efficacious by many successful cases.

The types of cases in which a patella ligament has been used have varied a great deal. Besides dislocation of the patella, it is used in certain derangements of the knee in which there is apparent looseness of the joint and a sensation of weakness when weight is applied to the partly flexed knee. A fascial ligament is useful to repair a complete or almost complete rupture of the patella tendon itself. In some cases the complete congenital dislocation of the patella was accompanied by a malposition of the quadriceps and it was necessary to transplant the quadriceps to a middle position on the anterior part of the leg in order to be able to replace the patella and later to insert a fascial ligament from the patella to the inner side of the tibia to insure a permanent position of the patella and to give stability to the knee. The ligamentous operation is done several months after the muscle transplantation.

One of my patients with a gunshot wound and complete rupture of the patella tendon itself had an almost complete disability of the knee. A broad fascial ligament was attached from the patella to the tibia with return of normal function.

OPERATION

The surgeon selects the position for the supporting fascial ligament. An incision is made above or below this line so that a flap may be turned back down to the fascia. The patella is tunneled obliquely from above downward and from without mesially. At about the middle of this tunnel a window is opened in the top. A similar tunnel is made in the tibia in approximately the same line and well to the mesial side of the tibia. The fascia on the mesial side of the tibia is heavy and strong and overlies a ridge of bone. Through this ridge a tunnel may be made easily with an osteotome instead of drilling as described for the patella. This prominent ridge at the side of the tibia simplifies the process of tunneling. While the osteotome is in place in the tunnel, a window is cut down to it through the overlying fascia as described above for the patella. The ligament in the patella is passed from the mesial side of the tunnel upward and the loose end, as it emerges from the outer side of the patella, is folded over the top of the patella and tucked in through the window, emerging below at the entrance to the tunnel on the mesial side of

*Deceased February 21, 1933.

Soutter—See "This Week's Issue," page 105.

In cases of weakness of the hamstrings, the tensor fasciae latae may be displaced backward along the course of the biceps femoris and attached medially to the biceps in the head of the fibula. This operation can be combined with a tenodesis of the biceps to the femur in cases of genu recurvatum.

TRANSPLANTATIONS AT THE HIP

There are two hip conditions which are very disabling—paralysis of the extensors and paralysis of the abductors. These may occur singly or in combination; when they occur together, the hip joint may dislocate.

FIG. 15. Shows sartorius and iliotibial bands sutured to ligamentum patella. Reflected aponeurosis and periosteal flaps of patella being sutured to the transplanted tendons.

In a pure abductor paralysis, the technique of Legg gives excellent results. Formerly Legg advocated inserting that part of the fascia lata just below the tensor into the lateral aspect of the femur about two or three inches below the tip of the trochanter. In many cases the results of this procedure were excellent; but, as the insertion of the tensor is a little forward of the center of gravity, the full abduction power of the tensor was not always obtained, so lately Legg has advocated transplanting the origin of the tensor backward in order that the line of pull may be in line with the long axis of the femur. This operation gives better results than the former operation; but the surgeon has to be very careful to avoid injuring the superior gluteal nerve, and the suturing of the upper portion of the tensor to its new position is a little difficult, since the line of suture is below the crest of the ilium and into fascia. The writer has modified Legg's technique in several instances with good results. The anterior border of the tensor is mobilized and the origin is divided along the crest of the ilium to a place just above the trochanter. The belly of the muscle is next freed from the deep structures and the whole muscle is turned backward on itself so that the anterior edge is sutured to the fascia behind the trochanter. In this way there will be a straight pull from the crest to the insertion of the iliotibial band. The upper edge is sutured to the underlying intact fascia.

Paralysis of the gluteus maximus gives a very disabling and unsightly limp and it also is very fatiguing to the patient. Seven years ago the writer described an operation to help relieve this paralysis. A large number of patients have submitted to this operation, many of them with double hip flexion contractures and with no power below the waist. All of them have been helped to a marked degree, but only a few have had complete relief from their disability. Several of these patients can get about the house without crutches. All of them can get out of chairs more easily. They can navigate stairs a great deal better, and the tremendous backward body thrust in walking has been relieved to a great extent in the majority of instances. In other words, there has been a definite improvement in function, which is very important in any crippling condition.

In the early operation, the iliotibial band was mobilized beginning at its attachment on the lateral aspect of the fibula, and extending upward to the muscle belly of the tensor fascia lata. This muscle was freed at its anterior and posterior aspects, and the iliotibial band was drawn through a drill hole made through the shaft of the femur opposite the lesser trochanter. The free end was then passed up over the gluteus maximus and sutured to the outer three-fourths of the erector spinae group. In a later development of this operation the free flap of fascia is passed under the muscle and periosteum two inches below the base of the trochanter, firmly sutured to this at the entrance anteriorly, and to the gluteus maximus tendon posteriorly. The new tendon is passed upward over the gluteus maximus and sutured to the erector spinae group, which has been previously mobilized in the following manner: the lower five or six inches of the erector spinae are exposed. The erector spinae muscle is split longitudinally in a line of cleavage about one-half inch lateral to the spinous processes of the last three or four lumbar vertebrae, extending down over the sacrum so as to free the tendinous portion of this muscle from the sacrum and posterior crest of the ilium. The whole muscle is separated along the lateral intermuscular septum and extending its whole depth down to the transverse processes and upward as far as the perforating dorsal nerves. The iliotibial band is secured to the under side of this muscular flap on either side and overlapping for about two to three inches. Many of these patients have had a severe lumbar lordosis, which in one case

had persisted for seventeen years. In all cases this severe lumbar lordosis entirely disappeared.

SUMMARY

1. Tendon transplantations at the ankle improve function if the cases are well selected and the operative technique is carefully followed out.
2. Tendon transplants combined with stabilizing operations are more useful because better stability results in better muscular control.
3. Many transplants may be done now which were formerly thought to be valueless.
4. Tendon transplants at the knee result in improved function and better security and stability of the knee.
5. The sartorius and tensor fasciae are the more ideal muscles to use; first, because of their anatomical situation, and secondly, the function of these two muscles is not so opposite as that of the hamstrings.
6. Transplants at the hip result in improved function and minimize an unsightly and troublesome gait.

A NEW OPERATION FOR SLIPPING PATELLA

BY ROBERT SOUTTER,* M.D., F.A.C.S.

SUCCESS of this operation in the long run depends on the technique and the mode of attachment which must stand all kinds of strains with the knee in any position. The points of attachment should be chosen to give the greatest mechanical advantage. The method here described has been used on a large number of adults and children with success.

This method was first used by the author in congenital dislocations of the patella at the Children's Hospital, Boston. Since then, both adults and children have been operated on by the same method. It is so much simpler than changing the angle of the condyle by cutting the femur and setting it at an angle, cutting away half of the patella ligament and transplanting half, reefing the capsule which is useless, and many elaborate methods which involve alteration in the existing tissues or opening the joint.

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the patella. This locks the fascia in place. Two catgut sutures are placed in the fascia. In a similar way the fascial ligament is tucked in on the outer side of the tibial tunnel. It emerges at the mesial side of the knee. The loose end is then brought out at the mesial side of the tibia (note illustration), folded over and tucked

FIG. I. Diagram showing the extra articular position of the ligament attached from the patella to the tibia.

FIG. II. Skeletal diagram showing the ligament entering the tunnel in the patella and in the tibia.

in through the window and emerges at the outer side of the tunnel. The two loose ends being then approximated, are passed through each other twice and sutured with catgut. This last procedure may be omitted but was done in all cases and makes for added strength. The detail of making the tunnel, of seeing that it is large enough for the folded fascia, will be mentioned below.

An incision is made over the outer side of the thigh of the good leg, or, of the affected leg, down to the fascia lata. A strip is removed 1¼" or 1½" broad. The necessary length is carefully measured so that it will reach twice the distance between the farther side of the tunnel in the tibia to the farther side of the tunnel in the patella. Before removing the strip any large amount of fat is scraped off. It is next folded so that it will be three thicknesses and about three-eighths of an inch broad. The ends are narrowed one-half for one-half inch so that they may readily start through the tunnels.

ALTERNATE METHOD

The ligament may be attached to the fascia over the patella and below to the fascia over the tibia, so long as the attachment is strong and holds the patella firmly. Another method has been used lifting a small portion of the bone from the tibia and a small portion of the bone on top of the patella so that the tunnel was practically made in the fascia, superficially under the bone. In very small children it is very easy to get through the cartilaginous patella and the soft bone of the tibia. A satisfactory tunnel where the patella is small is made under the fascia and a thin layer of cartilage. The bony attachment first described is to be preferred however, and in the long run is more reliable.

In drilling the patella in adults, one heavy silk suture is placed above and one below the patella in the firm fascial tissue close to it. These are used to steady the patella during the drilling. The patella is then held in place by pressure on the opposite side from the drill and by holding these sutures. Three small drills are used and each one is drilled through the patella separately. As the drill emerges from the opposite side of the patella, it is rotated several times so that it will be loose in its hole. It is then detached from the drill and left in position. A second drill is passed through parallel to this, a short distance from it and a third in like manner. When all three are in place they are withdrawn and a larger drill inserted in the same holes. After three large drills are passed through the bone, a small osteotome, which is not so thick as the diameter of the drill, nor so wide as the three drills, is gently passed through the patella. When the osteotome emerges from the other end of the tunnel, a window is removed from the top and middle of the tunnel down to the osteotome. The tunnel will be large enough to take several thicknesses of fascia lata easily and there will be no difficulty in tucking the end through as described above. The regular steel drills, number three and number five, are used. These steel drills may be bought at any hardware store and are standard sizes. They are very satisfactory but

the ends are not sharpened for use in bone. They can be sharpened to a point on a small emery wheel.

The wounds are closed in the usual way. A very small gauze dressing is put over the wound and about three thicknesses of sterile sheet wadding. A plaster is applied for three weeks. A large window is cut in the plaster over the knee. Icebags are put on either side over the sterile sheet wadding and the ice is renewed every hour for the first twenty-four hours. This adds a great deal to the comfort of the patient, although the swelling is not great if the tissues have been handled carefully.

Normal function is the rule and in time the previously overstretched muscles contract to their normal length and add strength to the knee maintaining the patella in position. Some of the patients had severe accidents several years after the operation. One man, while tobogganing, three years after the operation, caught his foot when going at high speed and injured his knee which was tremendously swollen. He was taken to a hospital in the mountains and put in bed for nearly two months. When I saw him the ligament was not disturbed by the accident and the following summer he was able to climb mountains and tramp seventy-five miles. His knee was as good as the other.

These operations have been very satisfactory in all cases and demonstrate the value of fascial ligaments from the patella to the tibia in restoring normal function and preventing dislocation of the patella.

TENSOR FASCIA FEMORIS TRANSPLANTATION IN CASES OF WEAKENED GLUTEUS MEDIUS

BY ARTHUR T. LEGG, M.D.*

IN January, 1923, the writer published the report of an operation to strengthen the abductor power of the thigh by transplanting the iliotibial band into the lateral side of the femur, in cases where the gluteus medius has been weakened by infantile paralysis.

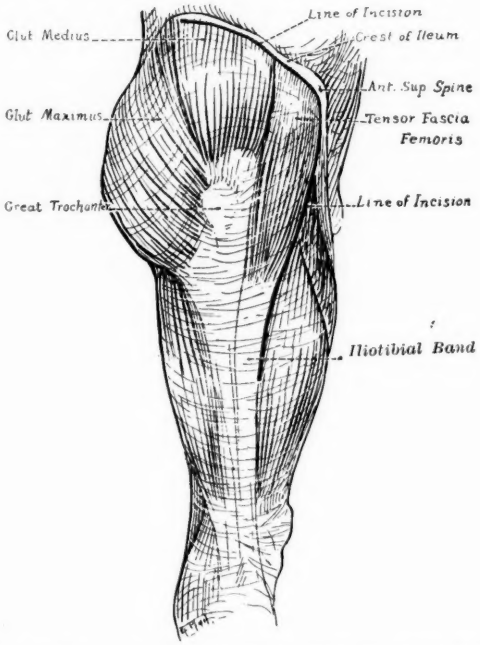

FIG. 1. Drawing showing normal anatomy about the hip with the line of the incision.

While these cases showed marked improvement in abductor power due to a more direct pull of the tensor fascia femoris on the femur, the muscle still had a flexor pull, since its origin is in front of the line of true abduction. Later, in 1926, after first doing this transplantation of the iliotibial band into the femur, a second stage was added, the origin of the tensor fascia femoris being transplanted backward along the crest of the ilium to directly above the great trochanter. This gave a straight pull in the line of true abduction.

The results from this operation were much better than those obtained by the original operation. The transplanted muscle now had a straight abductor pull between the crest of the ilium and the middle of the femur. Since the iliotibial band, however, representing the tendon of the tensor fascia femoris, normally extends down the whole thigh to the outer condyle of the tibia, the writer believed that a still better mechanical pull could be obtained by omitting the first stage of the operation in which the iliotibial band was inserted into the femur, and doing only the second stage where the origin is moved back to the center of the crest of the ilium in a line directly above the great trochanter.

This operation has been done since August, 1928, and up to the present time about twenty-five cases have been so operated upon with extremely gratifying results.

OPERATION

The incision is made along the crest of the ilium, starting a little behind the middle and going forward to the anterior superior spine, then downward along the inner border of the

*Legg—Assistant Professor of Orthopedic Surgery, Harvard University Medical School. For record and address of author see "This Week's Issue," page 105.

tensor fascia femoris to the mid-thigh. The skin and subcutaneous fat are reflected back, exposing the fascia lata. The anterior border of the tensor fascia femoris is now identified, and fascia between it and the sartorius is divided, the division being carried down along the anterior border of the tensor fascia femoris and continued down along the anterior border of the iliotibial band. This is necessary in order to allow the muscle and fascia to be set back.

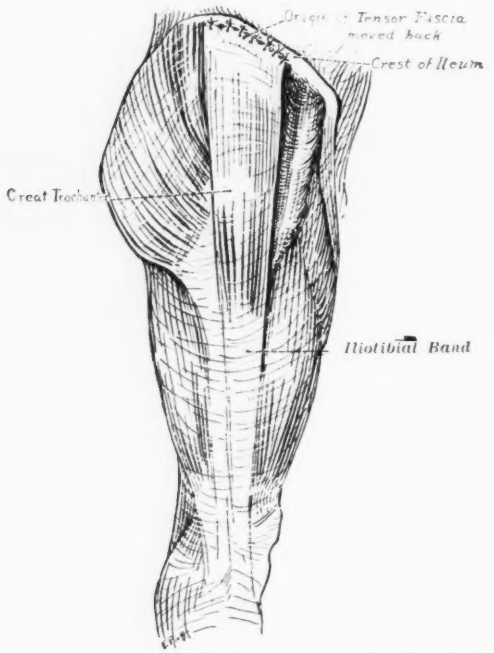

FIG. 2. Drawing showing origin of the Tensor Fascia Femoris moved back to the crest of the ilium.

The origin of the tensor is now freed from the anterior crest of the ilium, taking with it some of the cartilage of the crest. At this stage the fascia must be freed from the great trochanter anteriorly in order to get the muscle directly over the trochanter. The leg is now abducted about 35° and the origin of the muscle is sutured with number 18 silk to the crest, in a line directly above the great trochanter. The subcutaneous fat is then sutured with catgut, and the skin with black silk. After the dressing is applied, the leg is put into a bivalved plaster spica which has been made previously, to maintain the position of 35° abduction.

It has been my custom to dress the wound on the eighth or ninth day and to begin light muscle training at the end of twelve days. Muscle training is gradually increased and at the end of six weeks the patient is allowed to walk in an abduction splint, to insure holding the leg in abduction and to prevent too much strain being put on the transplanted muscle. The abduction splint is removed at the end of three months.

It should not be forgotten that habit plays a considerable part in any limp which has existed for some time, and that the patient will tend to revert to his original limp when the brace is discarded, unless emphasis is laid upon training in walking and balancing the body at that time.

There is little danger of destroying the nerve supply to the tensor fascia, as it comes from behind, through the superior gluteal, and the origin of the muscle is freed subperiosteally from the crest.

As was mentioned in the original paper, the tensor fascia femoris cannot take up the complete function of the gluteus medius when this muscle is completely paralyzed, but even in this event it will diminish the lateral sway of the body to a great extent. In cases with a fair amount of power in the gluteus medius, this transplantation will oftentimes eliminate the abductor limp.

CHANGES IN THE EPIPHYSIS—SECONDARY TO INFECTION*

BY JAMES WARREN SEVER, M.D.†

THE three principal infections, which cause changes in the epiphysis, and which I wish to call to your attention today, are tuberculosis, syphilis and osteomyelitis.

We, of the older generation, were brought up to depend on the x-ray diagnosis of bone lesions. We, in the past, have had definite word and roentgenological pictures presented to us, as typical of the above-named conditions. Yet as time goes on, we are required to furnish more and more clinical information to the radiologist in order that an approximate diagnosis may now be made. Yet today, after years of observation, we all are more and more unwilling to say definitely what the bone lesion presented in the x-ray really is. This holds true also, not infrequently, of the pathologist. The clinician, the radiologist and the pathologist, working together and pooling their observations, may come to an approximate diagnosis, but many times we all fail.

This is not an expression of failure, but is the result of an increased knowledge and study of bone pathology, from x-ray interpretations, and

*Read at the Annual Meeting of the New England Surgical Society at Hartford, Conn., October 1, 1932.

†Sever—Assistant Professor of Orthopedic Surgery, Harvard University Medical School. For record and address of author see "This Week's Issue," page 105.

a sensing of the fact that bone reactions are many times alike to different mechanical, chemical and infectious stimuli.

Lovett and Wolbach[1] have called attention to the fact that bone is a structure of very limited reaction to pathological conditions.

They state that there seem to be only three reactions possible in bone, (1) atrophy or diminution of lime content, (2) destruction of bone—local and general, (3) a formative process, characterized by formation of new bone, or a condensation of existing bone around a focus of disease.

CUT 1.*

CUT 2.

TUBERCULOSIS. NO. 1 AND 2.

No. 1 and No. 2; R. C., 19 mos. old. X-rays one month apart; showing destructive process in lower end of tibia; duration five weeks; diagnosed as osteomyelitis; Wassermann negative; bovine and human tuberculin tests positive. Died of tubercular meningitis; pathology proved to be tuberculosis.

They conclude that tuberculosis is largely destructive in character; that osteomyelitis is at first destructive and then formative; that syphilis has been considered the most purely formative of these three processes.

They believe that the diagnosis of infectious lesions of the bone would be simple, if each infectious agent always produced the same reaction.

To review now for a moment the so-called classical description of bone and epiphyseal changes as seen by the radiologist in tuberculosis, syphilis and osteomyelitis.

Tuberculosis: Bone atrophy; a destructive process generally beginning in the epiphysis; irregular erosion with puncture into the joint through the epiphysis. A generally destructive process, with no new bone formation as a rule; a cloudy, irregular epiphyseal line, and generally involvement of the joint surfaces. A condition supposed previously to begin in the epiphyseal line and work toward the joint. Multiple lesions uncommon.

NO. 3. L. L., aged 8 7/12 years; tuberculosis of knee involving Tibial Epiphysis; 4 years' duration; Wassermann negative; proved tuberculosis; June, 1931, had recurrence with swollen, painful knee. Leg was ½" longer than the non-affected one. Right knee.

NO. 4. M. G., aged 7 years; duration one month; proved tuberculous by biopsy; Wassermann negative. Two years later wound stayed healed and the legs were of equal length.
In this case one might well suspect strongly a localized osteomyelitis.

Syphilis: The process beginning in the epiphyseal end of the diaphysis, with marked periosteal thickening and so bone thickening; multiple lesions are common; an irregular epiphyseal line is formed and there may be sep-

*It is impossible to publish all the slides shown but certain ones are reproduced which show the points under discussion.—J. W. S.

aration of the epiphysis. Periostitis may occur early in the congenital form without much destruction of the epiphysis; with no involucrum and no sequestra.

Osteomyelitis: Marked destruction of normal bone follows as seen in x-ray. It begins with a focus in the diaphyseal end of the bone next to the epiphysis. Bone proliferation and

NO. 5. E. T. Osteomyelitis. Head of Tibia. Wassermann neg. T. B. tests ++. Guinea Pig neg. for T. B. Pathology—Staphylococcus aureus—14 mos. duration. Note involvement of head of tibia, and epiphyseal line.

NO. 6. R. K., aged 12½ years. Bony ankylosis of the right knee, with question of sequestrum at the lower end of the femur; six years' duration. Still has slight discharging sinus. Note complete bony fusion of both tibial and femoral epiphyses.

sequestration are present with joint involvement and separation, and destruction of the epiphysis. Extensive destruction of bone cortex takes place.

These descriptions are not unlike. They all present pictures of bone and epiphyseal involvement as well as joint invasions of varying degree. How shall we tell them apart in an x-ray?

What happens to the epiphysis as a result of these various types of infection? This can be answered only by the knowledge of two factors, namely, the amount of involvement, or destruction of the epiphyseal cartilage plate, which is

NO. 7. D., 14 years old; osteomyelitis of the knee with bony ankylosis; five years' duration; bony ankylosis of the knee with involvement of the patella and femoral epiphysis.

between the metaphysis and the epiphysis; and by time.

To review the literature of epiphyseal changes, as the result of infection, or trauma, is too lengthy a process to go into here, but certain facts should be noted. These facts have been determined by various investigators as the re-

NO. 8. March 9, 1927. T. F., aged 23 mos. Staphylococcus osteomyelitis upper end of the femur. Wassermann and tuberculin tests negative; pathological report osteomyelitis.

sult of laboratory experiment and clinical experience.

The proper growth of the epiphysis depends to a certain extent on the future mechanism of the joint; the opposite is likewise true. The growth of the epiphysis may be influenced by chemical and mechanical means. Meisenbach[2] showed in 1910, by animal experimentation, and the injection of various chemical solutions into the epiphyseal line, that the epiphyseal line

could be stimulated or destroyed. He found that a formalin solution was the best chemical for epiphyseal stimulation. In his animals he was able to rapidly stimulate the epiphysis to overgrowth at first, but later this chemical irritant showed a destructive effect on the zone of provisional calcification, with retardation of growth and eventual shortening occurring. Therefore, he concluded that apparently chemical stimulation, if it is carried to the point of destruction of the cartilage plate between the metaphysis and the epiphysis, results in loss of longitudinal growth.

NO. 9. September 14, 1927. Shows increased destruction of upper end of femur.
Same at No. 6.

Haas[3] has shown that the most active and important elements anywhere for longitudinal growth are located in the columns of cartilage cells in the so-called epiphyseal cartilage plate previously spoken of. If these cartilage cells are destroyed by chemical or mechanical means, or their circulation is interfered with, loss of growth inevitably occurs. Haas[3] has also shown that the longitudinal growth of bone is practically dependent upon the integrity of the vascular supply to the epiphysis. In other words, the maintenance of the normal longitudinal growth of bone is dependent upon a sufficient blood supply to the region of the epiphyseal cartilage plate. Interference with this blood supply is undoubtedly one of the causes of the considerable shortening one sees in many of these cases of infected epiphysis. To be sure, at first, many of these infections result in an early stimulation of this cartilage plate, either as the result of an increased blood supply, or as the result of irritative effects of toxins developed as the result of the infection. With the death of these epiphyseal cartilage cells, growth eventually stops, and a short limb results, with inevitable asymmetry of the joint. If the normal blood supply to the epiphyseal cartilage plate or to the epiphysis itself can be maintained, particularly through the nutrient artery, the loss of growth, as the result of damage to the cartilage cells, may be less marked than when the nutrient artery is injured or occluded. When actual trauma or infection takes place in the region of the epiphyseal line, or when the injury is to the cartilage plate, the greater is the growth disturbance. There is always a definite relation in the degree of destruction of the cartilage column, and the eventual loss of growth. These effects have been definitely determined by Haas in his animal experimentations.

You are all familiar with the various endeavors in the past, to stimulate epiphyseal growth by means of ivory pegs and other foreign substances which have been introduced deliberately into the epiphyseal line or adjacent to it in an endeavor to increase the length of the short limb.

Bohlman[4] has shown as the result of his animal experimentations in 1929 that, as the result of using all kinds of material, none of the materials he used produced any increased growth in length, as a matter of fact, marked shortening occurred, and he does not feel that the use of any such material is warranted in any way, as they interfere definitely with the normal growth of the cartilage plate.

In order to overcome deficiency in growth, it has been suggested many times that complete transplantation of an epiphysis might be possible to take the place of the injured or infected one which has ceased growing.

NO. 10. April 22, 1929. Shows regeneration of bone and healing. With some involvement of epiphyseal line.
Same as 6 and 7.

None of the laboratory experiments on animals, by observers in this country, have shown that this was possible. The reason for the failures is undoubtedly due to the marked interference in the blood supply, not only from the nutrient artery, but from the cortical branches, as the result of the operative trauma necessary for such a procedure. On the other hand, there are two observers who claim success with this

method. Todyo[5] of Japan reported several years ago, as the result of experiments on animals, that most of the epiphyseal cartilage and the epiphyseal line, following transplantation, have lived and carried on active proliferation and ossification.

This is rather against the result of other investigators. The other observer, Straub[6] of Honolulu, reports a case in 1929 of a successful transplantation of a piece of the diaphysis of the tibia, with about one half of the epiphysis attached to it, from one leg to the other. In his case, which he followed for many years, he found that the epiphysis survived, grew and continued to function perfectly normally. I believe his is rather an unique experience, and again is rather against the experience of Haas[7] in his report on the results of his animal experiments in 1915. Haas believes that the epiphyseal cartilage loses its power to function after transplantation; that the epiphyseal cartilage should be considered a very vulnerable tissue, and that its viability is directly dependent upon its blood supply, which is wholly interfered with as the result of such transplantation, and he concluded that, longitudinal growth, therefore, ceased as the result of such a procedure involving the cartilage plate.

You are all familiar with the traumatic epiphyseal separations which take place, and you have probably wondered why, in many of these instances, so little eventual dysfunction has occurred.

Epiphyseal separation at the hip, reduced by either closed or open methods, often leads to marked limitation of motion in that joint, with flexion deformity. Other epiphyseal separations I believe make good immediate recoveries, but if followed for several years will show many changes in growth and asymmetry, as the result of the original trauma.

The separation of the epiphysis as the result of such an injury usually takes place in a line between the metaphysis and the so-called epiphyseal cartilage plate, which is between it and the epiphysis itself, and as a result of this anatomical arrangement, there may be no gross interference with the circulation, or of the viability of the epiphyseal cartilage columns. The epiphyseal cartilage plate, not only receives its blood supply from the nutrient artery, but some observers state that its most important supply is from the cortical branches, which find their way in from the cortex along the epiphyseal line, and so are distributed to the whole of the epiphysis.

You can see from this very brief review that the integrity of the epiphysis and the maintenance of longitudinal growth depend wholly on the lack of injury or disturbance to the epiphyseal cartilage plate, and its blood supply, and particularly the vertical column of cartilage cells, which lies directly underneath the epiphyseal line. Infections which disturb these cells, as a whole, or in part, eventually result in a loss of longitudinal growth and asymmetry of the joint.

This preliminary review is merely to introduce the main subject of this paper to you, namely, the changes in the epiphysis due to infection. The slides which I am about to show you, I will explain in the short time available. That many of them show similar lesions, as the result of different infections, you will admit. That a definite diagnosis could be made, without laboratory confirmation, is hardly to be expected.

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CONGENITAL TORTICOLLIS

Review of the Pathological Aspects

BY HENRY J. FITZ SIMMONS, M.D.*

THE pathology of Congenital Torticollis has apparently had only superficial interest to the writers on this subject. The clinical observers have accepted only those views with which they have been of like opinion. Unfortunately, opinions frequently have been expressions without any substantiation or proof.

The writer's interest has been of some years' duration. In a paper published in 1915[1] a group of clinical cases were studied. In 1927 a study made but not published, showed little change in the accepted treatment of this condition, but a

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disturbing return to the careless confusion seen some years before. Congenital torticollis was apparently diagnosed when acquired torticollis would have been the more correct designation.

This paper deals only with the literature since the last study, 1927, and is particularly designed to draw attention to the pathological aspects. It is hoped thereby to clarify the confusion and again direct attention to more rational treatment.

The attached partial bibliography on the subject is fairly conclusive, and should offer to one wishing to do so, a list of articles some of which are well worth reading. The writer has not had an opportunity to read them all, but has read many and is quoting now from two articles which he feels are of particular interest and benefit.

D. Stewart Middleton[2] states in some work which is particularly well done, and from which the writer has quoted extensively, that any attempt to clarify the question of torticollis must deal with the following points: (I) the previous theories which have been advanced to explain these points, and a short discussion of the weak features of such theories. (II) The relationship, if any, between congenital torticollis and the so-called "sternomastoid tumour" of infancy; the morbid anatomy of the muscle in sternomastoid tumour and torticollis. (III) The etiology of the sternomastoid tumour; experimental reproduction of similar changes in muscle in animals. (IV) The etiology of various changes in the skeleton associated with the presence of torticollis.

1. *The Hereditary Theory.* This theory, supported by the observations of Jeannel[3], Pfeiffer, Fisher, and Joachimsthal[4] the author believes, with most writers on this subject, cannot be maintained. Cases of torticollis occurring in parent and offspring are so infrequent as to render it certain that they are merely coincidences.

2. *The Congenital Theory.* The view that the contracture like clubfoot or hare-lip, is due to some abnormality in the development of the foetus, or to some abnormal pressure on the foetus by the walls of the uterus, might be held to account for those rare cases where an infant is born with a fully developed torticollis. In the vast majority of cases, however, the wry-neck does not appear until months or years after birth. It would also be difficult to explain by this theory why the contracture should be completely limited to the sternomastoid muscle in all cases except old-standing adult ones where secondary adaptive changes in surrounding muscles and fasciae take place. Finally, a congenital theory cannot account for the fibrosis present in the contracted muscle. In the above statement one sees the shadow of confusion, and the need of defining congenital torticollis.

3. *The Theory of Birth Injury.* Stromeyer, who formulated this theory, holds that the sternomastoid muscle is ruptured during labour giving rise to the "sternomastoid tumour", which is in reality a hematoma, and that the contracture following the organization of this hematoma is responsible for the wry-neck. This is the theory which Dr. Stewart Middleton believes commonly receives support today. However, it is hard to explain birth trauma without subsequent torticollis and congenital torticollis without any semblance of obstetrical injury.

He does show later, however, that this theory contains a grain of truth. It realizes that there is an intimate relationship between the presence of a sternomastoid tumour and the subsequent development of a wry-neck, and also that both these conditions have some connection with long and difficult labours. He feels that the theory errs in attributing the sternomastoid tumour to the presence of a hematoma following rupture of the muscle during labour. At no time does the swelling show any of the characteristics of a hematoma, being from its inception firm, hard, and cartilaginous to touch, and never fluctuating or showing any signs of bruising. It does not appear immediately after birth. It affects in most cases the greater part of the muscle instead of a localized portion of it, and on microscopical examination it shows no appearance which can in any way suggest a hematoma. Finally, true hematomata produced experimentally by tearing a muscle leave no trace of contracture behind them and in no way resemble sternomastoid tumours (Witzel[5]).

4. The theory of infective myositis following upon contusion of the sternomastoid muscle at birth. This was based upon the microscopical appearances of the excised muscle. It has been shown by Kempf[6], however, that there is no foundation for the belief that there is any infection present. He comes to the conclusion that the microscopical appearances seen are those of the repair of a hemorrhagic infarct, a statement which is, as we shall see later, entirely true.

5. *The Nervous Theory.* Few authorities support this theory. Golding Bird incriminated a cerebral lesion in intra-uterine life in which Gallavardin and Savy[7] observed atrophy of the anterior-horn cells in the cervical cord in an adult case of torticollis. It is probable that these changes are secondary to the destruction of muscle, and are analogous to the atrophy of anterior-horn cells which follows upon the amputation of a limb.

6. *The Ischemic Theory.* First suggested by Mikulicz, this view was supported when Nové-Josserand and Viannay[8] undertook a detailed investigation of the circulatory system of the sternomastoid muscle, and showed the possi-

bility of interference with the blood-supply of the muscle taking place during labour. This theory marks the most modern and most accurate work on the subject, and, as we hope to show, it comes very close to the truth. It recognizes that the muscular fibrosis seen in torticollis is essentially a similar process to that seen in Volkmann's ischemic contracture of the flexor muscles of the forearm. It errs in adopting too whole-heartedly the belief which was then (1906) widely accepted, that Volkmann's contracture results from arterial deprivation to the flexor muscle, a belief which has always been and remains today completely unsupported by any sound evidence, clinical, pathological, or experimental.

RELATIONSHIP BETWEEN THE STERNOMASTOID TUMOUR AND TORTICOLLIS: MORBID ANATOMY

It is commonly stated in textbooks that though torticollis, when it appears early in life, may follow directly the absorption of a sternomastoid tumour, yet the majority of cases appearing later in childhood have not been in any way connected with such a condition. Both types of torticollis present the same clinical and pathological appearances, and it would therefore appear rational to expect that all cases are attributable to a common cause.

Let us consider the main facts with regard to sternomastoid tumour and torticollis.

The sternomastoid tumour of infancy. The tumour appears usually from one to two weeks after birth, the commonest time being about ten days. It is, so far as his (D. Stewart Middleton's) observations go, never present at birth. It is first noticed as a spindle-shaped swelling occupying the position of one sternomastoid muscle. Occasionally it affects only the sternal head of the muscle, but very frequently both sternal and clavicular heads are thickened. The small uppermost portion of the muscle close to the mastoid attachment is, however, seldom if ever affected. The enlarged muscle is hard to the touch, and gives a typical "cartilaginous" sensation to the examining fingers which I have never felt elsewhere apart from the experimentally produced tumour which will be described later.

If a child is suffering from such a tumour and is kept under observation it is found that the tumour remains more or less stationary for from two to three months, and then is gradually absorbed, disappearing in from four to six months after birth.

It is not uncommon for such a child to exhibit a mild degree of torticollis while the tumour is at its height, but this is transient and is of the nature of an acute torticollis. As the tumour is tender to touch, the child holds the head to one side in order to relax the affected muscle so far as possible. On the other hand, in some cases with a well-marked tumour, a true torticollis commences at about four months of age when the tumour is diminishing in size, the head being apparently drawn over to one side coincidently with the absorption of the tumour.

Morbid Anatomy. If the sternomastoid tumour is excised and examined, it appears to the naked eye to be composed entirely of glistening fibrous tissue, and presents an appearance not unlike a soft fibroma. On microscopical examination it is found to consist of young and cellular fibrous tissue containing here and there the remnants of the original muscle fibers. Many of these fibres show an absence of nuclei, vacuolation, and are undergoing degeneration.

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Morbid Anatomy. If the contracted sternal or clavicular head from a case of fully developed torticollis in a child of, say, eight years of age is excised and examined the microscopical picture can only be interpreted as representing the terminal stage of a sternomastoid tumour. No degenerated muscle or young fibrous tissue is observed, but instead, swathes of adult noncellular fibrous tissue are seen, with, scattered amongst it all, collections of muscle fibres and, though smaller than normal and varying somewhat in size and outline, living healthy fibres that bear none of the stigmata of degeneration. The microscopical appearances in themselves form convincing evidence that torticollis represents the end-result of a sternomastoid tumour; and, on investigating 36 cases of torticollis it was found that 25 gave a definite history of a sternomastoid tumour shortly after birth which underwent absorption and was followed at varying periods up to some five or six years later by the development of torticollis. The cases in which no history of a tumour was obtained were mainly in older children, and are in all probability accounted for by changes in guardianship or the failure of a parent's memory to recall a trifling ailment of a passing character affecting one child of a family some six or ten years before. It is, in fact, an occasional experience in the out-patient department to discover a sternomastoid tumour which has remained unnoticed in a stout baby.

Reason for late development: One point remains to be dealt with at this stage. How is it, if torticollis results from the fibrosis consequent upon the absorption of the sternomastoid tumour, that the wry-neck may not be noticed for years afterwards, in the majority of cases appearing about the age of four? A little thought will make this clear. The neck of an infant is short, and it is not until about the age of four that the individual commences to grow from the proportions of a "child" into those of the "small boy or girl". The whole bodily type of

a child changes at this period. Prominent among the changes which occur is an increased growth in height, and this is especially marked in the relative increase in length of the neck, which assumes almost adult proportions. Suppose a child at this age to have suffered during infancy from a sternomastoid tumour which has become absorbed and left behind it a degree of residual fibrosis which was not sufficient to cause the immediate development of a torticollis. Such a muscle is crippled as regards the function of growth in comparison with its normal neighbor on the opposite side. The muscle substance on the affected side is partly replaced by an abnormal tissue which will not elongate with growth to the same extent as the normal muscle. When the growth period arrives, therefore, the fibrosed muscle lags behind in its elongation and becomes relatively shortened producing the classical deformity.

Production of Venous Obstruction in Labour. It is difficult at first to explain the mechanism by which a venous obstruction can occur during labour, though a satisfying anatomical explanation is forthcoming in the counterpart of this condition, Volkmann's contracture of the flexor muscles of the forearm (Middleton[9-10]). The sternomastoid muscle is derived from the three different muscular segments. Muscle fibres derived from these segments flow into the neck and unite to form the three portions of the muscle, each portion retaining unaltered its own original arterial supply and consequently its own venous return.

The upper mastoid portion, which is very much shorter than one is generally led to believe, is situated at the mastoid extremity of the muscle, and is supplied by the superior sternomastoid artery derived from the occipital artery. This artery is short and runs a transverse course. The venous return from this portion of the muscle appears to travel through short trunks which penetrate the deep aspect of the muscle and join the large veins beneath. It is difficult to conceive of any mechanism by which these veins could become obstructed, and in fact a careful examination of sternomastoid tumours shows that this small mastoid portion of the muscle appears seldom, if ever, to be affected, and this portion is not the seat of fibrosis in torticollis.

The sternal head of the muscle receives its arterial supply through the middle sternomastoid artery, a branch of the superior thyroid artery. This branch takes origin from its parent trunk a short distance above the upper border of the omohyoid muscle. The vascular bundle is seen and divided in every case of excision of glands in the neck as it runs backwards and downwards parallel to the upper border of the omohyoid muscle. On reaching the external head of the muscle, it does not at once enter it. It passes beneath this head, appearing in the interval between the sternal and clavicular heads. It then courses downwards along the posterior border of the sternal head giving branches forwards to that head as it goes, but sending no branches to the clavicular head. The venous return from the sternal head appears to follow the artery of supply, and eventually terminates in the venous plexus formed by the junction of the superior thyroid and lingual veins with the common facial vein.

The clavicular head derives its arterial supply from the inferior sternomastoid artery, a small branch coming either direct from the thyreocervical trunk or from one of its branches, usually from the transverse scapular artery. The branch runs upwards upon the deep aspect of the muscle supplying branches to it. The exact path of the venous return from this head is a little uncertain. Probably much of the blood is returned by a vein accompanying the artery of supply, but Mr. Saunders, of the Anatomical Department of the University of Edinburgh, has observed small venous trunks issuing from the surface of the muscle to join the external jugular vein.

Nové-Josserand and Viannay[8], investigating the circulation of the sternomastoid muscle in the stillborn foetus, found that these three systems of blood-supply were independent, each supplying its own portion of the muscle and failing to communicate with each other or surrounding arteries. If this is so, it follows, of course, that the venous systems are also independent. Impressed with the theory of arterial deprivation, they proceeded to show that the arteries to the sternal and clavicular heads can be occluded in the foetus if the head is placed in a position of extreme rotation such as occurs during labour, the tense sternomastoid muscle in this portion being capable of compressing the vessels of supply where they pass beneath it. If, under experimental conditions, the middle and inferior sternomastoid arteries can be closed to injection by this means, it is not too much to suppose that the thin-walled veins may be shut off during labour without at the same time interrupting the arterial supply, thus reproducing, temporarily at all events, the condition obtaining in the experimental muscle after venous ligation. It is perhaps hardly necessary to emphasize the fact that such a conclusion must remain at present in the realm of pure theory, but it appears that some such hypothesis offers the only explanation of the origin of the sternomastoid tumour and congenital torticollis which is not seriously at variance with known clinical and experimental facts.

Another point remains to be cleared up in this connection. If sternomastoid tumour results from venous blockage in the positions of rotation assumed by the foetal head during the

passage of the pelvis minor, one would expect that such tumours would commonly occur following upon complicated labours, or normal labours which had been unduly prolonged, having therefore an unusually high incidence in primiparous cases. A survey of our series of cases gives us the following information: Out of 64 cases where the place occupied by the child in the family was known, 43 were the first children born to the mother. Of the remaining 21 who were not born of primiparae, 16 were classified as either "difficult prolonged labours", "cross-births" or "breech presentations". Out of 83 cases in which the history of the labour was known, only 14 were described as normal, 46 were "difficult" or prolonged, 15 were "breech presentations", and eight were "cross-births".

It is known that obstetrical paralysis of the Erb-Duchenne type almost invariably follows on a difficult delivery succeeding a prolonged and troublesome labour, being especially common after breech presentations. One would expect to find a high proportion of sternomastoid tumours associated with such cases. The histories of 54 cases were reviewed, of Erb's paralysis which had been under observation during the main period (from birth to three months) in which tumours might be found. The series is consecutive except that all cases which were not seen till they were over three months of age are excluded. Out of 54 such cases during the last five years, no fewer than 11—that is to say about one case in five had exhibited a sternomastoid tumour.

Jepson[11] has shown that a venous blockage which has been complete for some hours is capable of setting up a process of fibrosis which continues even after the venous obstruction has been relieved. This might quite conceivably occur during labour, but nevertheless one feels that there must be some other factor at work which is capable of rendering such a temporary venous blockage permanent. It seems likely, judging from the appearance of the veins within a few minutes of experimental venous ligation, that thrombosis might well occur in the stagnant blood occupying the greatly engorged venous trunks in the muscle, and, in point of the fact, such thrombosis can be observed, under the microscope in the earlier stages of the process. If the whole venous tree of, for instance, the sternal head of the sternomastoid muscle were blocked for an hour or two during labour, thrombosis occurring in parts of the tree would obviously render the interference with the venous return from portions of the muscle permanent, with resulting fibrosis. This would explain the fact that the fibrosis of torticollis is classically patchy in character with intervening areas of normal muscle. I have carefully examined many sections of muscle from sternomastoid tumours and cases of torticollis in an endeavour to find the remains of these thrombosed veins. One might expect that they would be difficult to find amongst masses of fibrous tissue months or years after the date of thrombosis, and further, small nerve-trunks on section must be carefully distinguished from them. Dr. Stewart Middleton says that he has been unable definitely to demonstrate such veins either to his own satisfaction or to that of Dr. James Davidson, Pathologist to the Royal Infirmary of Edinburgh, to whom he acknowledges indebtedness for much help on this point. His conclusions are as definite as is his entire paper. By quoting from it so extensively the writer of this review acknowledges his great debt and appreciation.

D. Rossi[16] makes a very complete study of a child extracted by cesarean section in which torticollis was present. He also clearly states that the various theories on the etiopathogenesis of torticollis may be summarized under two headings; congenital and traumatic-obstetrical. It would seem that in speaking of "congenital" torticollis, its origin has already been accepted; but this term is also used to indicate other types; that which is acquired as a result of trauma, the spastic functional ocular type due to nystagmus, and the type due to occipitalization of the atlas. He refers to the theory advanced by Petersen (going back to Hippocrates), which connects the etiopathogenesis with the intrauterine life of the fetus, and explains the mechanism as an abnormal pressure exerted by the uterine walls on the product of conception, which assumes a permanent faulty attitude, with rotation and flexion of the neck. This causes an abnormal shortening of the distance between the points of insertion of the sternocleidomastoid, and later constitutes caput obstipum. Petersen, after numerous experiments on animals and on the basis of clinical observations, concluded that the faulty intrauterine position of the fetus is related to amniotic adhesions, to oligohydramnios, and to various other conditions which also cause other disturbances in form.

The second theory maintains that the affection is acquired after delivery due to violent or improper obstetrical manipulations, due to application of forceps to the neck of the fetus. This theory was contested, first because forceps are not applied to the neck, then because torticollis was recorded even before there were any forceps. To support the theory, it has been suggested that the laceration of the sternocleidomastoid is caused by traction exercised in disengaging the head in podalic or shoulder presentations, and also in cephalic presentations with difficult delivery. It has been demonstrated, experimentally and clinically, that a lacerated muscle does not become shortened.

CONCLUSIONS

1. In all cases congenital torticollis results from fibrosis in the sternomastoid muscle consequent upon the development and absorption of the so-called "sternomastoid tumour" of infancy.
2. Sternomastoid tumour, and therefore torticollis, results from a temporary acute venous constriction in the muscle, this obstruction taking place during labour.
3. It is probable that such a temporary venous obstruction has been rendered permanent by patchy intravascular clotting in the obstructed venous tree.

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COUNTERSINKING THE ASTRAGALUS IN PARALYTIC FEET

BY ALBERT HOWELL BREWSTER, M.D.*

STABILIZATION of the ankle in paralytic feet has been of profound interest to surgeons for years. Before describing the following operation I wish to acknowledge my indebtedness to Dr. Michael Hoke of Atlanta, Georgia, because had I not been acquainted with his fundamental procedure, this idea probably would not have presented itself. The following procedure was first done in the summer of 1927. Since then over fifty of these cases have been treated by this technique. The operation has not been described previously, because it seemed wise to let sufficient time elapse to be sure the results warranted such a procedure.

Countersinking the astragalus in paralytic feet has for its purpose:

First—Lateral stability of the foot.
Secondly—Limitation of dorsiflexion.
Thirdly—Limitation of plantar flexion.
Fourthly—After the above is accomplished, the foot appears normal and is functionally good.

Technique: An incision is made on the lateral side of the foot from the head of the astragalus convex downward to the posterior superior surface of the os calcis. (Fig. 1.)

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Through this incision the skin and subcutaneous tissues are divided. The peroneal tendons, peroneus longus and brevis are exposed, and retracted from the field of operation. The fossa between the inferior surface of the astragalus and superior surface of the os calcis is de-

FIG. 1. Incision.

nuded of tissue. Beginning at the junction of the neck and body of the astragalus, its inferior surface is removed in a plane parallel to the sole of the foot. This removes the cartilage of the astragalus which forms the superior surface of the joint between the astragalus and the os calcis.

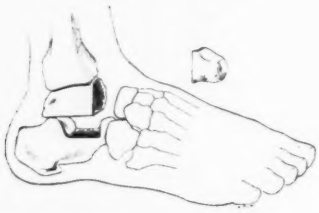

FIG. 2. Diagram to show first stage of operation to countersink the astragalus.

At this point it is well to point out certain anatomical findings in the os calcis which are of much importance in countersinking the astragalus into the os calcis. Just posterior to the calcaneocuboid joint there is a bony elevation on the anterior superior surface of the os calcis. There is a second bony elevation on the posteri-

FIG. 3. Diagram shows fitting of the anterior end of astragalus in os calcis. Line posterior indicates where more bone is to be removed from posterior part of os calcis and astragalus.

or superior surface of the os calcis, which partially takes part in the formation of the joint between the astragalus and the os calcis. (Fig. 1.)

Now taking advantage of the bony elevation on the anterior superior surface of the os calcis above mentioned, a chisel is driven through the os calcis at right angles to the sole of the foot, and just posterior to the cartilage of the calcaneocuboid joint, from the lateral side to the medial. At right angles to this the superior

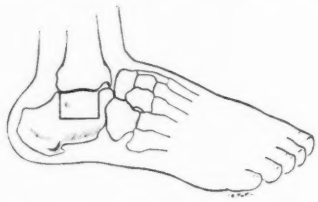

FIG. 4. Astragalus countersunk into the os calcis.

surface of the os calcis is removed parallel to the sole of the foot anteroposteriorly, until all the cartilage of the os calcis taking part in the joint between the astragalus and os calcis is removed.

The head and neck of the astragalus are next

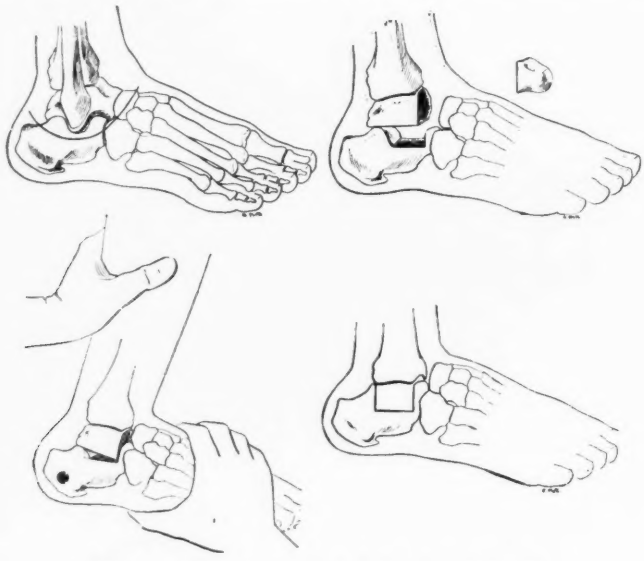

FIG. 5. Shows figures 1, 2, 3 and 4 in sequence.

removed at right angles to the sole of the foot, so as to remove part of the cartilage on the superior surface of the astragalus, which forms the joint between the tibia and the astragalus. Two right angles have now been made by the above described procedure:

First—In the astragalus anteriorly, by the plane of its inferior surface, and the plane at right angles to this produced by the removal of the head and neck in the manner previously described.

Secondly—In the os calcis anteriorly, by the plane of its superior surface, and the plane at right angles to this produced by removing bone from the bony prominence on the anterior superior surface of the os calcis previously mentioned. (Fig. 2.)

After the foot is displaced backward it is then determined at what point the posterior part of the astragalus and os calcis are to be removed. (Fig. 3.) The posterior part of the astragalus is removed at right angles to the sole of the foot. This plane makes a right angle with the inferior surface of the astragalus. Next, as much of the bony prominence on the posterior superior surface of the os calcis is removed at right angles to the sole of the foot as is necessary. This plane makes a right angle with the superior surface of the os calcis. The two right angles are made to fit as the two anterior ones were, and the countersinking of the astragalus is accomplished. (Fig. 4.)

It must be mentioned that the cartilage of the scaphoid, which takes part in the formation of the joint with the head of the astragalus, is removed. If the foot is sufficiently displaced backward it is not necessary to replace any part of the head and neck of the astragalus. (Figs. 4 and 5.)

This procedure gives lateral stability, and it limits plantar flexion and dorsiflexion to the degree desired. The foot appears nearly normal, and is functionally good.

FIG. 6. X-ray of left foot after the astragalus has been countersunk.

FIG. 7. X-ray of right foot after astragalus has been countersunk.

FIG. 8. X-ray of right foot after the astragalus has been countersunk.

FIG. 10. Shows a bad postoperative result, because range of motion in plantar flexion and dorsiflexion is too great for a flail foot.

FIG. 9. Photograph of foot after operation, and its range of motion, both in plantar and dorsiflexion.

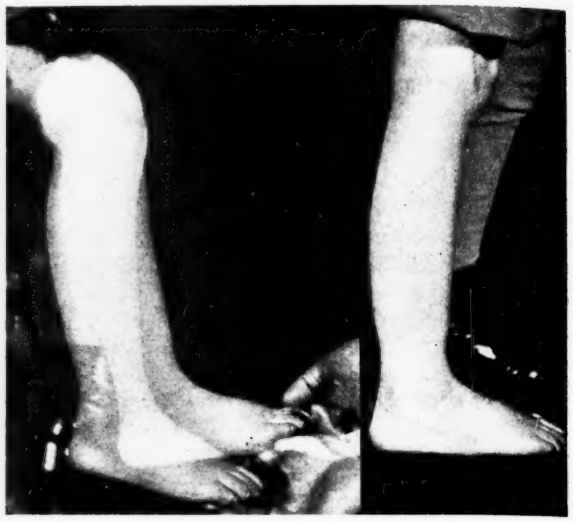

FIG. 11. Photograph of foot after operation, and its range of motion both in plantar and dorsiflexion.

"FLEXION DEFORMITY" OF THE HIP AND THE LATERAL INTRAMUSCULAR SEPTUM

BY S. M. FITCHET, M.D., F.A.C.S.*

HIPPOCRATES was the originator of so much in surgery that the present-day surgeon often finds himself disappointed and embarrassed when claiming an original procedure. The underlying principles of an old method and a new procedure are often the same and have formed the basis of discussion ever since their enunciation by the recording Priest of Aesculapius.

We suppose hip flexion and hip abduction deformities have existed almost since the beginning of man, and we shall not quarrel with anyone who discovers that the methods herein proposed are ancient or submodern.

This paper lays no claim to originality but merely stresses the simplicity of adequate treatment of so-called "hip flexion deformity", which is so frequently noted as a resistant deformity complicating poliomyelitis. The first essential is adequate recognition of the structures at fault. This recognition appears to be lacking in many cases, and accounts for the failures in obtaining cures.

Yount[1] has made a very careful analysis of the deformity of hip flexion contracture and the frequently associated deformity of knocked knees. Much of this excellent study will be summarized in order to emphasize the points in treatment which will be discussed later.

Most surgeons dealing with this deformity fail to recognize the fact that the most important element is the hip abduction rather than a true hip flexion.

Soutter did some most excellent pioneer work and very accurately summarized the situation when he wrote, discussing these common deformities of poliomyelitis, that contractures of the soft tissues of the hip may hold the thigh flexed and abducted, and involve a shortening of more or less all the soft tissues on the outer and anterior aspect of the region about the hip. He noted, too, that there was frequently a compensatory lordosis.

The treatment up to the time of Soutter's[2] careful study and well-thought-out operation was stretching with or without tenotomies. Frequently manipulation and stretching were of little use. Tenotomizing of the tendons and tensor fascia lata and even myotomies were resorted to.

Soutter sought for and devised an operation which would overcome this deformity in the most difficult cases. With his operation the deformity was corrected with but little bleeding and without doing myotomies. A distinct advantage as he pointed out was that the operation presented no great difficulties to the average bone and joint surgeon.

There is no question whatever as to the efficiency of this procedure in correcting the deformities. The operation, however, is often moderately shocking in the debilitated and extremely paralyzed. It would seem, therefore, to be a procedure to be reserved for those dif-

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ficult cases in which simpler methods might not succeed. There are modifications of the Soutter operation, such as Campbell's[3] and Albee's[4], and to these modifications the same rule applies. Speed[5], an associate of Campbell, reported very satisfactory end results in 100 cases in which a modified Soutter operation was employed. In each of these cases there were 20 degrees or more of flexion deformity. He called attention to the influence of abduction in the flexion deformities. The 100 cases included the following:

85 Poliomyelitis
7 Spastics
4 Infectious Arthritis
4 Tuberculosis

The ilio-psoas muscle is the great flexor of the hip. It is difficult to believe, however, that section of this muscle would ever be justifiable in attempting to correct flexion deformities of the hips complicating cases of poliomyelitis.

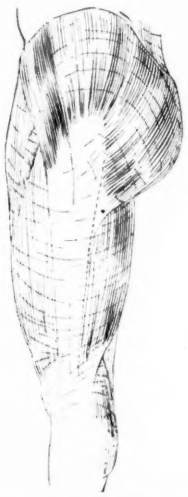

FIG. 1. Dotted line represents the position of the lateral intramuscular septum connecting the posterior edge of the ilio-tibial band to the femur.

One rarely, if ever, sees a true, uncomplicated flexion deformity of the hip. If the hip is abducted, the flexion disappears and extension of the hip or even hyperextension may readily be obtained. The deformity is really an *abduction* deformity of the hip, and myotomy of the ilio-psoas would avail little in correcting the deformity.

The tensor fascia femoris and a large portion of the gluteus maximus muscle insert into the fascia lata and continue down the thigh jointly as the ilio-tibial band inserting mainly into the tuberosity of the tibia. These are the structures chiefly involved in the abduction deformity. The sartorius, a knee flexor, is rarely important unless there is very great deformity at the hip, and then only secondarily.

The fascia lata is attached to the femur throughout its length by means of intramuscular septa which arise from it. The lateral intramuscular septum is an extremely strong structure uniting the posterior edge of the ilio-tibial band firmly to the femur. This septum lies between the vastus externus and the short head of the biceps femoris.

The tensor fascia femoris is not only a tensor of the fascia, but is also an abductor of the thigh. The gluteus maximus inserting into the fascia lata is also a tensor of this fascia as well as an extensor of the hip. The gluteus maximus is therefore important in abduction deformity of the hip. The tensor fascia femoris and the

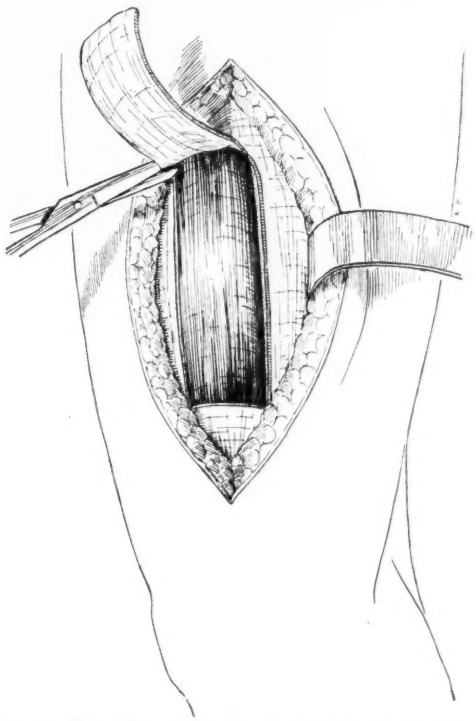

FIG. 2. Removing block of ilio-tibial band (fascia lata). The intramuscular septum lies just posterior to the vastus externus.

gluteus maximus acting through their common insertion, the ilio-tibial band, are definitely abductors of the thigh. The fact that the tensor fascia femoris and the gluteus maximus are also abductors of the thigh by virtue of the strong attachment of the whole of the fascia lata to the femur by the lateral intramuscular septum is not generally recognized.

I do not know who first discovered the importance of the lateral intramuscular septum in so-called hip flexion deformity, but the first mention of it in the literature was made by Yount. Soutter had called attention to the fact that the anterior and lateral structures of the thigh were responsible for the deformity and directed surgical procedures against them for correcting the deformity. Silver recognized the importance of the ilio-tibial band. Forbes[6] noted the attachment of the ilio-tibial band to the femur by an intramuscular septum in de-

scribing a transplant of this structure to patella. He states that Spitzy in 1914 was the first to describe a transplant of the fascia lata to the patella. Silver, Yount and their associates did

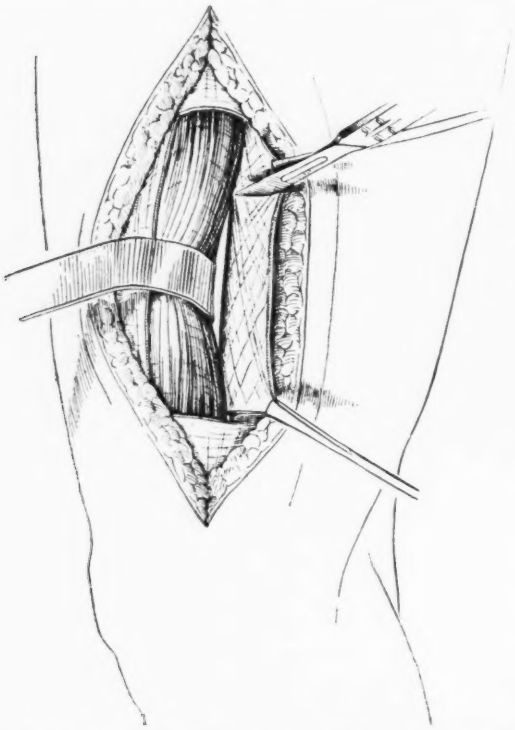

FIG. 3. Section of the intramuscular septum. (Block of fascia lata, ilio-tibial band, has been removed.)

simple, subcutaneous fasciotomy of the ilio-tibial band and, quoting from Yount, "except in those cases in which marked deformities are of long standing and of marked degree, better

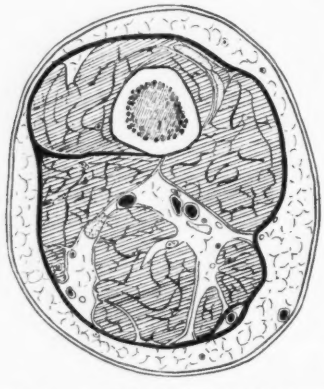

FIG. 4. Cross section of thigh showing intramuscular septum continuous with the fascia lata and connecting it to the femur with the vastus externus anterior, and the short head of the biceps femoris posterior.

final results are obtainable with division of the ilio-tibial band at the knee than with any other method of treatment".

This has not been our experience. We feel that in all cases the intramuscular septum must be sectioned if a complete and thorough operation is to be done and to minimize the likelihood of recurrence of the deformity. Yount recognized the importance of the septum, but did not sufficiently emphasize it, so that what appeared as a surgical procedure of merit lost favor with some operators because it was incompletely done.

Our procedure is as follows:

An incision about three inches long is made over the ilio-tibial band extending about two inches above the proximal level of the patella and about one inch below, and carried down to the ilio-tibial band.

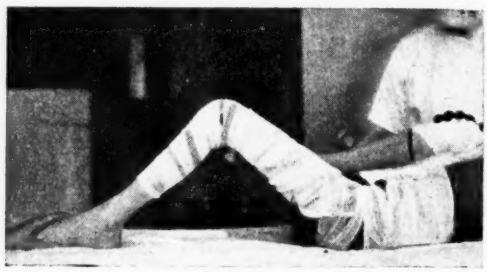

FIG. 5. 40° deform.

The ilio-tibial band is isolated. If there is a weak quadriceps, the band is sectioned at its attachment, drawn out through a small wound higher up on the lateral aspect of the thigh and brought down with a tendon carrier to be inserted in the patella. The ilio-tibial band may be utilized in paralysis of the hip adductors as

FIG. 6. 15 minutes later. The intramuscular septum has been sectioned.

Mayer[7] suggests, where it is carried to the pubis and made fast. Or it may be used by attaching it to the erector spinae muscles for weak glutei, as done by Ober[8]. If it is not desired to use the ilio-tibial band, a one to three inch segment is removed, and the fascia lata sectioned anteriorly and posteriorly sufficiently to relieve tension. This is never done subcutaneously.

Very close to the posterior margin of the ilio-tibial band and structurally continuous with it, the tight intramuscular septum is identified lying between the vastus externus muscle anteriorly and the biceps posteriorly. A section of this septum, one to three inches long, is removed. In cases of marked deformity the septum may be stripped one-half to two-thirds or even all the way up the femur. For stripping the fascia and intramuscular septum, we have devised an

instrument as illustrated (Fig. 9) and find it very useful, but not at all essential.

Section of the ilio-tibial band and the intramuscular septum usually permits the hip to drop into complete extension in the adducted position. The importance of the division of the septum as well as the ilio-tibial band is appreciated by observing the widening of the gap in

FIG. 7. 35° deform.

FIG. 8. 15 minutes later. The intramuscular septum has been sectioned.

the divided band as soon as the septum is released. The operation is completed by mere closure of the wound.

We have had no unhappy results from the stretching of nerves and vessels following the sudden and complete extension after prolonged hip abduction deformity. It is well, however,

FIG. 9.

to remember this possibility and use due caution.

The sartorius has never proved troublesome; it rarely is a factor in this deformity. It may always be stretched or lengthened should the knee flexion deformity be marked and secondarily influence the hip.

We have purposely omitted any discussion of knee flexion deformity and knock-knee deformity which is so often associated with abduction deformity of the hip. The subject has been admirably discussed and analyzed by Yount. We also omit any discussion of the frequently associated deformity of lordosis.

It is worth while mentioning other conditions in which the above operation has been found useful.

In treating congenital dislocation of the hip, one is often discouraged at the difficulty encountered in getting back adduction after prolonged treatment in abduction. Section of the ilio-tibial band and the lateral intramuscular septum has been found useful. It has also been of some value in selected cases of spina bifida and spastic paralyses.

In some cases of congenital abduction and flexion deformity of the hips, the operation has been of use as a preliminary procedure and before the hip joint capsule has been approached.

Its greatest field of usefulness, however, will be in the treatment of the frequent and troublesome deformity of abduction-flexion in cases of poliomyelitis.

Postoperative care depends entirely on other factors in the case. If knee flexion is to be corrected, or unstable feet to be stabilized, or any other procedure to be carried out, it should be done without other caution than retaining the corrected position of the hips until the patient becomes ambulatory.

We have found this operation a definitive and worth-while procedure in the abduction-flexion deformity of the hips (flexion deformity of the hips) in infantile paralysis. We give credit to Soutter and Yount for focusing our attention on the lateral structures of the thigh in this deformity. We suggest that this procedure does not supersede Soutter's excellent operation, but that Soutter's procedure be reserved for selected cases where such a maneuver is desirable because of the extreme nature of the deformity. We emphasize the importance of an open operation, complete section of the ilio-tibial band, section of the anterior and part of the posterior fascia lata, and, most important, careful and complete section of that very restraining insertion of the abductors of the hip, i.e., of the lateral intramuscular septum.

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FOOT STABILIZATION

A Review of Fifty-Two Operations

BY ROBERT H. MORRIS, M.D.*

THE problem of foot stabilization has been examined and dealt with very effectively, so that today the surgeon may choose from several methods that one which best suits the case in hand. It is not the purpose of this paper to discuss the relative merits of the different types of operations in use, but rather to show the results of fifty-two foot stabilizing operations. To show why some are perfect and to suggest some of the possible causes for the failure to attain perfection in others. A few years ago Dr. Hoke suggested the following requirements of a stabilized foot.

1. To look natural in shoes.
2. To be so stabile that it will not turn laterally on the long axis of the foot when the patient is standing or walking.
3. To be so stabile that in the natural or nearly natural attitude it does not need a brace to hold it so.
4. To look natural when barefoot or nearly so to prevent any gross deformity.

Perhaps these four requirements may be summed up by good function and good appearance.

Good function is undoubtedly the first consideration, because any operation is a failure which produces a good-looking foot that won't function. If, however, good appearance is added to good function the result is surely close to the ideal. In this series every good-looking foot is a good functioning foot. There are, however, thirty-one operated feet which have good function, but owing to some deformity were not good-looking. In view of these results the question to be answered is, would these thirty-one operated feet have functioned better without this deformity. The answer seems to be strongly in the affirmative. In other words, the better the appearance the better the function.

A perfect postoperative foot must of necessity vary from the normal in appearance in two ways. One, the foot will look short. Two, the relationship of the malleoli and astragalus to the os calcis will be slightly anterior to the normal. It is true that in some cases an equinus or a slight varus or valgus may be a helpful compensating factor for disability higher up in the leg. However, the feet having so-called compensatory deformity are not included with the perfect feet.

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Some writers hold the opinion that a foot may be stabilized by a judicious transplantation of tendons alone, claiming for this method the best possible results both as to function and appearance. There are others who believe that an astragalectomy is the only means of procuring a stabilized foot. There are still others who believe that an arthrodesis of one or more joints with or without a tendon transplantation the method of choice.

The operations considered in this series are of the latter variety, three types of arthrodesing operations having been used, and a large percentage of these combined with tendon transplantation.

1. Subastragalar arthrodesis; in which the articulation between the astragalus and calcaneus is fused.
2. Hoke stabilization; in which the articulation between the astragalus and calcaneus is fused, the head and neck of the astragalus removed, the proximal surface of the scaphoid denuded of cartilage, a portion of the head and neck of the astragalus denuded of cartilage and inserted between the scaphoid and the astragalus.
3. Triple arthrodesis; in which the articulations between the astragalus and calcaneus, astragalus and scaphoid, calcaneus and cuboid are fused.

AGE

The average age at time of operation is seven years. This represents the general consensus of opinion as to the suitable and safe age to stabilize a foot. One child was operated on at the age of three and a half years and the result shows this to be a complete failure as there is no fusion and the foot is wholly unstable. The period of time between the onset of the disease and the operation varies from one and a half years to ten years, the interim in the great majority of cases being three years.

POSTOPERATIVE DEFORMITIES

The postoperative deformities have been considered with great care and perhaps with too great severity, so that a casual glance at the chart gives the impression that the results are very unsatisfactory. However, a closer inspection shows the reverse to be true.

The degree of deformity was classified according to the amount of deviation from the normal alignment of the foot.

0 to 5 degrees variation, perfect postoperative foot.
5 to 10 degrees variation, slight (s. l.) deformity.
10 to 15 degrees variation, plus (+) deformity.
15 to 20 degrees variation, two plus (++) deformity.
20 degrees variation and over, three plus (+++) deformity.

Sixteen (30%) of all operated feet show no postoperative deformity and are considered perfect feet both as regards appearance and function. These perfect feet all have good backward displacement, solid bony fusion, and perfect or almost perfect muscle balance laterally and anteroposteriorly. Twenty-four (46%) have good function, but it is believed that without the associated deformity, which is slight, they will function better. Eight (15%) show plus (+) deformity. Four (9%) show two plus (++) deformity or greater, and reoperation is recommended for these. Absence of backward displacement failure of complete fusion and imperfect muscle balance account for all the deformities.

BACKWARD DISPLACEMENT

To attain backward displacement of a foot there must be a change in the normal relationship between the astragalus and calcaneus. This change may perhaps be described as a moving forward of the astragalus and malleoli to a position somewhat forward of the normal; so that the thrust of the body weight transmitted through the lower leg and astragalus to the os calcis is somewhat anterior to the normal vertical axis. To produce backward displacement in a sub-astragalar arthrodesis is mechanically impossible, for in this type of operation there can only be a lowering or tilting of the astragalus. The chart shows that this type of operation gives the poorest results as to appearance, there being postoperative deformities in every case. In the two other types of operation considered in this series backward displacement can be attained very easily, but not so easily maintained, because there is always a tendency for the astragalus to slide backward due to the contraction of the posterior ligaments of the ankle joint and this sliding back of the astragalus is always accompanied by a dropping of the forefoot into equinus. The maintenance of backward displacement is most important because with the astragalus and malleoli placed forward the foot is set in the best possible position for solid fusion between the astragalus and calcaneus and between the astragalus and the fore-foot; the former helping to control lateral mobility of the posterior part of the foot and the latter helping to control the inversion or eversion of the foot as well as helping to prevent fore-foot equinus. This forward position of the astragalus and malleoli also produces blocking of the foot in extension (dorsiflexion); a factor most important in a foot that has poor calf muscles. If close contact between the astragalus and fore-foot is not maintained only a slight amount of muscle imbalance is necessary to produce a deformity; whereas if contact is maintained slight muscle imbalance has no appreciable effect on the stability of the fore-foot and consequently deformity is not likely to occur.

There are two methods used which assure maintenance of backward displacement—

1. Postoperative repositioning of the foot as advocated by Dr. Hoke when he devised his operation for foot stabilization.
2. Counter-sinking the astragalus into the os calcis as devised by Dr. A. H. Brewster in his modification of Dr. Hoke's procedure.

FUSION

Clinically, solid fusion has been obtained in ninety per cent of the cases. X-ray shows only eighty per cent solidly fused. This means that ten per cent have fibrous union so solid that manipulation cannot produce lateral mobility. Ten per cent of the total operated feet show complete failure to fuse both clinically and by x-ray. This failure to obtain fusion is due to

1. Insufficient removal of cartilage at time of operation.
2. Inadequate immobilization following the operation.

X-ray study shows that in some cases the bone regeneration which took place between the arthrodesed joints is extremely uneven and abnormally great. In some cases the neck of the astragalus has regenerated to almost twice its normal size and shows marked torsion. This torsion is invariably associated with eversion (valgus). It is felt that this peculiar regeneration and twisting of the neck is due to the force of muscle pull and the thrust of the body weight on a poorly stabilized fore-foot.

MUSCLE TRANSPLANTATION

The method of estimating muscle power as shown in the accompanying chart is that in use at the Children's Hospital.

Good, (G) muscle acts against gravity and some resistance.
Fair, (F) muscle acts against gravity.
Poor, (P) muscle acts but not against gravity.
Trace, (T) very slight contraction.

There are some who doubt the value of transplanting tendons of the foot with the hope of their having any real balancing power in their new line of pull. The results of the thirty-two tendon transplants associated with arthrodesis of one or more joints in this series, show with-

TRIPLE ARTHRODESIS

No.	Age: Onset of Disease	Age: At Operation	Age: At Present	Deformity	Result: Clinical: Pain	Result: Clinical: Deformity	Result: Clinical: Fusion	Result: Clinical: *Ba-Disp	Result: X-Ray: Fusion	Result: X-Ray: *Ba-Disp
1	2/12	7	11	Valgus-Flail	No	Valgus sl.—Equinus sl.	Yes	Yes	Yes	Yes
6	4	6	11	Valgus (sl.)	No	Equinus (sl.)	Yes	No	Yes	No
7	1	6	10	Calcaneo-Valgus	No	Calcaneus (sl.)	Yes	No	*S. A. only	No
9	1/2	10	14	Equino-Varus	No	None	Yes	Yes	Yes	Yes
11	2 1/2	5	11	Equino-Varus	No	Valgus ++	Yes	No	Yes	No
12	5	9	13	Flail Cavus	No	Equinus sl.	Yes	No	Yes	No
13	2 1/2	6 1/2	11 1/2	Equino-Varus	No	None	Yes	Yes	Yes	Yes
15	3 9/12	5 9/12	7 9/12	Calcaneo-Valgus	No	Calcaneus sl.	Yes	Yes	*S. A. only	Yes
18A	2 6/12	6	11	Valgus +	No	Rigid Valgus +	Yes	No	Yes	No
21	10 1/2	12	13	Calcaneo-Valgus ++	No	None	Yes	Yes	Yes	Yes
22	1	7	10	Valgus	No	None	Yes	Yes	Yes	Yes
27	2	4 1/2	10	Equino-Varus	No	Equinus (sl.)	Yes	No	Yes	No
29	2	7	9	Calcaneo-Valgus	No	Equino-Varus sl.	Yes	No	Yes	No
31	3/12	6	8 1/2	Flail Valgus	No	None	Yes	Yes	Yes	Yes
32	4	6 1/2	8 1/2	Valgus	No	Calcaneus sl.	Yes	Yes	Yes	No
38	1	10	16	Equino-Varus	No	Equinus sl.	No	No	No	No
38A	1	10	16	Varus	No	Varus sl.	Yes	No	Yes	No
39A	2	9	12	Valgus	No	Valgus (Rigid)	Yes	Yes	Yes	Yes
40A	6	9 1/2	12	Equino-Varus	No	None	Yes	Yes	Yes	Yes

*Ba-Disp = Backward Displacement. S. A. = Sub Astragalar.

MUSCLE EXAMINATION—TENDON TRANSPLANT

No.	Gast.	A. T.	P. T.	Per.	E. D. L.	E. P. H.	F. L. D.	F. B.	F. L. H.		
1	F	O	O	P	O	O	P	G	P		
6	G	P	P	G	F	P	G	G	G	Peroneals to 2nd Metatarsal	Transplant working.
7	P	G	P	G	G	G	F	G	G	Posterior Tibial and Peroneals—to Os Calcis	Transplant not working.
9	G	O	O	O	O	O	T	G	G	E. P. H. to 1st Metatarsal	Transplant not working.
11	G	P	F	P	P	F	G	N	G		
12	O	O	O	O	O	O	O	O	O	Peroneus Longus to 1st Cuneiform	
13	P	T	P	O	O	O	P	P	P		
15	F	G	O	G	N	G	O	G	N		
18A	G	P	F	N	G	N	N	N	G	Peroneus Longus to 1st Cuneiform	Transplant not working.
21	P	T	F	G	F	F	F	F	F	Peroneus Longus to Os Calcis	Transplant working.
22	F	P	O	G	F	P	F	G	G	Peroneus Brevis to Os Calcis	Transplant working.
27	G	T	P	P	P	F	G	N	G		
29	F	T	P	P	P	P	P	P	P		
31	P	O	T	T	O	O	O	O	O		
32	F	P	P	G	F	G	F	P	G	Peroneus Longus to 1st Metatarsal	Transplant working.
38	N	O	T	O	O	O	P	F	F		
38A	G	N	O	F	N	N	N	N	N		
39A	N	P	T	N	N	G	G	N	N	Peroneus Longus to 1st Metatarsal	Transplant working.
40A	N	G	F	G	F	F	F	G	G	Anterior Tibial to 3rd Metatarsal	Transplant working.

39A	N	P	T	N	N	G	G	N	N	Peroneus Longus to 1st Metatarsal — Transplant working.
40A	N	G	F	G	F	F	F	G	G	Anterior Tibial to 3rd Metatarsal — Transplant working.

HOKE STABILIZATION

No.	Age: Onset of Disease	Age: At Operation	Age: At Present	Deformity	Result: Clinical: Pain	Result: Clinical: Deformity	Result: Clinical: Fusion	Result: Clinical: *Ba-Disp	Result: X-Ray: Fusion	Result: X-Ray: *Ba-Disp
4	1 1/3	5	11	Calcaneo-Valgus	No	Calcaneus sl.	Yes	Yes	Yes	Yes
4A	1 1/3	5	11	Valgus	No	None	Yes	Yes	Yes	Yes
5	4 1/2	11 1/3	16	Equino-Varus	No	Equino-Varus	Yes	No	Yes	No
8	4	6	12	Valgus	No	None	Yes	Yes	Yes	Yes
10	1	5	12	Calcaneo-Valgus	No	Varus-Cavus +	Yes	No	*Only A. S.	No
17	4	5 7/12	12	Calcaneo-Valgus	No	Varus sl.	Yes	No	*Only A. S.	No
19	2	5 1/2	12	Calcaneo-Valgus and Cavus	No	None	Yes	Yes	Yes	Yes
20	2	9 1/2	12 1/2	Calcaneo-Valgus	No	None	Yes	Yes	Yes	Yes
23	6/12	7	10	Equino-Varus	No	None	Yes	Yes	Yes	Yes
25	4	7	10	Equinus +++ Varus +	No	Varus sl.	Yes	No	Yes	No
28	4	8	12	Equino-Varus	No	None	Yes	Yes	Yes	Yes
34	3	6 1/2	8 1/2	Calcaneo-Valgus and Cavus	No	Varus +	Yes	No	Yes	No
36	1 1/2	9 1/2	12 1/2	Equino-Varus	No	Equinus sl. Varus +	*Only A. S.	Yes	*Only A. S.	Yes
37	3 1/2	6 1/2	8 1/2	Calcaneo and Cavus	No	Valgus +++	No	No	No	No
39	2	10 8/12	16	Valgus	No	None	Yes	Yes	Yes	Yes
40	4 8/12	6	12	Equino-Varus	No	Varus +	No	No	No	No
41	4 1/2	7	8 1/2	Valgus	No	None	Yes	Yes	Yes	Yes
43	6	9	10	Calcaneo-Valgus	No	None	Yes	Yes	Yes	Yes
45	1 1/2	4 1/2	6	Calcaneus and Valgus	No	None	Yes	Yes	Yes	Yes

*Ba-Disp = Backward Displacement. A. S. = Astragalar Scaphoid.

MUSCLE EXAMINATION—TENDON TRANSPLANT

No.	Gast.	A.T.	P.T.	Per.	E.D.L.	E.P.H.	F.L.D.	F.B.	F.L.H.		
4	T	P	T	F	F	F	P	P	P		
4A	G	T	T	T	G	F	F	F	F		
5	F	O	P	O	O	O	P	G	G	Tenotomy Posterior Tibial	
8	G	T	P	N	G	G	N	N	N	Peroneus Longus to 2nd Cuneiform	Transplant working.
10	O	N	N	N	G	N	N	N	N	Posterior Tibial, Peroneus Longus } to Os Calcis	Transplant working.
17	P	N	G	F	F	G	P	G	P	Posterior Tibial, Peroneus Longus } to Os Calcis	Transplant working.
19	F	G	T	N	N	G	N	N	N		
20	G	N	N	G	N	G	G	G	G	Posterior Tibial, Peroneus Longus } to Os Calcis	Transplant working.
23	F	P	T	O	F	F	G	G	G	E. P. H. to 3rd Metatarsal Posterior Tibial to Os Calcis	Transplant working.
25	F	T	P	O	O	O	F	G	F		
28	G	G	N	T	O	O	G	G	G	Anterior Tibial to Mid-Dorsum	Transplant working.
34	P	G	P	G	G	G	F	P	F	Peroneus Longus and Brevis, Posterior Tibial, F. L. D. } to Os Calcis	Transplants working.
36	G	G	O	O	F	G	F	G	G	Anterior Tibial to Mid-Dorsum	Transplant working.
37	F	N	G	N	N	N	N	N	N	Posterior Tibial, Peroneus Longus } to Os Calcis	Transplant working.
39	F	T	O	T	O	O	G	G	G		
40	G	F	T	F	F	F	T	G	F		
41	F	T	O	T	O	O	F	F	F		
43	F	N	G	N	F	G	N	N	P	Posterior Tibial, Peroneus Longus } to Os Calcis	Transplant working.
45	F	O	T	N	N	N	G	G	P	Peroneus Longus to 1st Metatarsal Flexor Digitorum Longus to Os Calcis	Transplant working.

SUBASTRAGALAR ARTHRODESIS

No.	Age: Onset of Disease	Age: At Operation	Age: At Present	Deformity	Result: Clinical: Pain	Result: Clinical: Deformity	Result: Clinical: Fusion	Result: Clinical: *Ba-Disp	Result: X-Ray: Fusion	Result: X-Ray: *Ba-Disp
2	6/12	10 1/2	14	Equino-Varus	No	Equino-Varus sl.	Yes	No	Yes	No
3	2	7	11	Valgus	No	Varus sl.	Yes	No	Yes	No
10A	1	10	12	Varus Cavus	No	Valgus. Calcaneo-Cavus sl.	Yes	No	Yes	No
14	3	5 1/12	7	Calcaneus	No	Valgus sl.	Yes	No	No	No
16	1	3 1/2	5 1/2	Calcaneo-Valgus	No	Valgus sl.	No	No	No	No
18	3 1/2	5 1/2	11 1/2	Calcaneo-Valgus	No	Equino-Valgus sl.	Yes	No	Yes	No
22A	1	5	7	Valgus	No	Valgus ++	No	No	No	No
24	6/12	6 1/2	8 1/2	Valgus +++	No	Valgus +	Yes	No	No	No
26	5	6 1/2	9 1/2	Valgus-Cavus	No	Valgus sl.	Yes	No	Yes	No
33	2 3/12	5 8/12	9	Valgus	No	Valgus +++	Yes	No	Yes	No
33A	2 3/12	9	10	Valgus +++	No	Valgus +	Yes	No	Yes	No
35	3	5	7	Calcaneo-Valgus	No	Equinus +	Yes	No	No	No
42	1 1/2	4 1/2	6	Valgus	No	Equinus +	Yes	No	Yes	No
44	3 1/2	6 1/2	8	Calcaneus	No	Valgus sl.	Yes	No	Yes	No

*Ba-Disp = Backward Displacement.

MUSCLE EXAMINATION—TENDON TRANSPLANT

No.	Gast.	A. T.	P. T.	Per.	E. D. L.	E. P. H.	F. L. D.	F. B.	F. L. H.		
2	G	O	P	O	T	T	F	G	G		
3	G	P	G	G	G	N	G	G	G	Peroneus Longus to Mid-Dorsum	Transplant working
10A	G	G	G	G	T	T	G	G	G		
14	F	G	P	G	G	G	F	F	F	Posterior Tibial, Peroneus Longus } to Os Calcis	Transplant working
16	P	G	F	G	G	N	N	N	N	Anterior Tibial, Peroneus Longus } to Os Calcis	Transplant working
18	F	O	T	G	F	G	F	F	G	Peroneus Longus to Tendon Achilles	Transplant working
22A	F	T	T	G	F	G	G	G	G	E. P. H. to 1st Cuneiform Peroneus Longus to Os Calcis	E. P. G. not working Peroneus Longus working
24	F	P	O	F	F	F	G	G	P	Peroneus Longus to 1st Metatarsal	Transplant working
26	G	O	N	N	N	N	N	N	N	Peroneus Longus to 1st Cuneiform	Transplant working
33	F	T	T	F	P	P	F	F	F		
33A	F	O	O	O	O	O	O	O	O		
35	P	G	P	N	P	F	G	G	F	Peroneus Longus, F. L. D., Post Tibial } to Os Calcis	Transplant working
42	G	O	T	P	O	O	P	G	G	Peroneus Longus, Flexor Longus Digitorum } to Os Calcis	Transplant working
44	F	N	G	N	G	N	G	G	G	Peroneus Longus, Posterior Tibial } to Os Calcis	Transplant working

out question that such a procedure is eminently successful. It is shown that the anterior and posterior tibial muscles as well as the peroneals can be used either as dorsi-flexors of the foot or placed in the os calcis to act as plantar-flexors. In only three cases is it impossible to demonstrate muscle action in the transplanted tendon.

It is perhaps less interesting but nevertheless most gratifying to find that not a single patient complains of pain. In every case but one, both parent and child were greatly pleased with the result of the operation regardless of how we estimated the function and appearance.

CONCLUSIONS

1. A good-looking foot is a good functioning foot.
2. A perfect postoperative foot must have
   (a) Good backward displacement,
   (b) Solid fusion,
   (c) Perfect or nearly perfect muscle balance.
3. Failure to maintain backward displacement is the greatest single cause of postoperative deformity.
4. Slight muscle imbalance is of no consequence in a well-fused and backwardly displaced foot. Whereas, with non fusion, slight muscle imbalance will cause marked deformity with sometimes great overgrowth of new bone.
5. That arthrodesis combined with tendon transplantation is an ideal method for procuring good function and good appearance in a stabilized foot.

TUBERCULOSIS IN INFANCY AND CHILDHOOD

A Statistical Study

BY MIRIAM G. KATZEFF, M.D.*

"GENTLEMEN, I wish to call your attention to the fact that today this orthopaedic ward does not contain a single case of tuberculosis. This is a very different picture from that of not so many years ago when it was not a difficult task to select tuberculosis cases for student demonstration."

This statement, heard so often on orthopaedic ward rounds, has stimulated a statistical study of tuberculosis cases in the Children's Hospital of Boston.

The study includes the total number of patients admitted annually to the hospital with a diagnosis of tuberculosis from the year 1884 to 1931, and a comparative statistical study of the orthopaedic and non-orthopaedic forms.

TABLE I

Year	Tuberculosis Cases	Orthopaedic Cases	Non-Orthopaedic Cases
1884	83	75	8
1885	102	98	4
1886	10	10	0
1887	61	55	6
1888	63	51	12
1889	80	68	12
1890	59	51	8
1891	58	42	16
1892	208	161	47
1893	199	172	27
1894	263	217	46
1895	161	138	23
1896	86	67	19
1897	94	66	28
1898	367	298	69
1899	.....	.....	.....
1900	.....	.....	.....
1901	554	459	95
1902	556	457	99
1903	.....	.....	.....
1904	.....	.....	.....
1905	.....	.....	.....
1906	.....	.....	.....
1907	.....	.....	.....
1908	.....	.....	.....
1909	.....	.....	.....
1910	.....	.....	.....
1911	335	179	156
1912	418	303	115
1913	458	385	73
1914	495	326	169
1915	410	272	138
1916	505	313	192
1917	428	242	186
1918	439	290	149
1919	430	273	157
1920	351	215	136
1921	360	231	129
1922	333	199	134
1923	257	145	112
1924	262	127	135
1925	273	116	157
1926	274	85	189
1927	216	88	128
1928	267	66	201
1929	294	86	208
1930	278	61	217

In the early years of the hospital records, 1884 to 1891 inclusive, cases of tuberculosis were not great in number, but in 1892 there was a sudden increase which continued until 1894. For the next three years, 1895 to 1897 inclusive, the number diminished but there were still more cases annually than during the first eight years. In 1898 there was again a marked increase in the cases of tuberculosis admitted to the hospital reaching a high peak in 1901 and 1902.

*Katzeff—Junior Assistant Surgeon, Orthopedic Department, Children's Hospital. For record and address of author see "This Week's Issue," page 105.

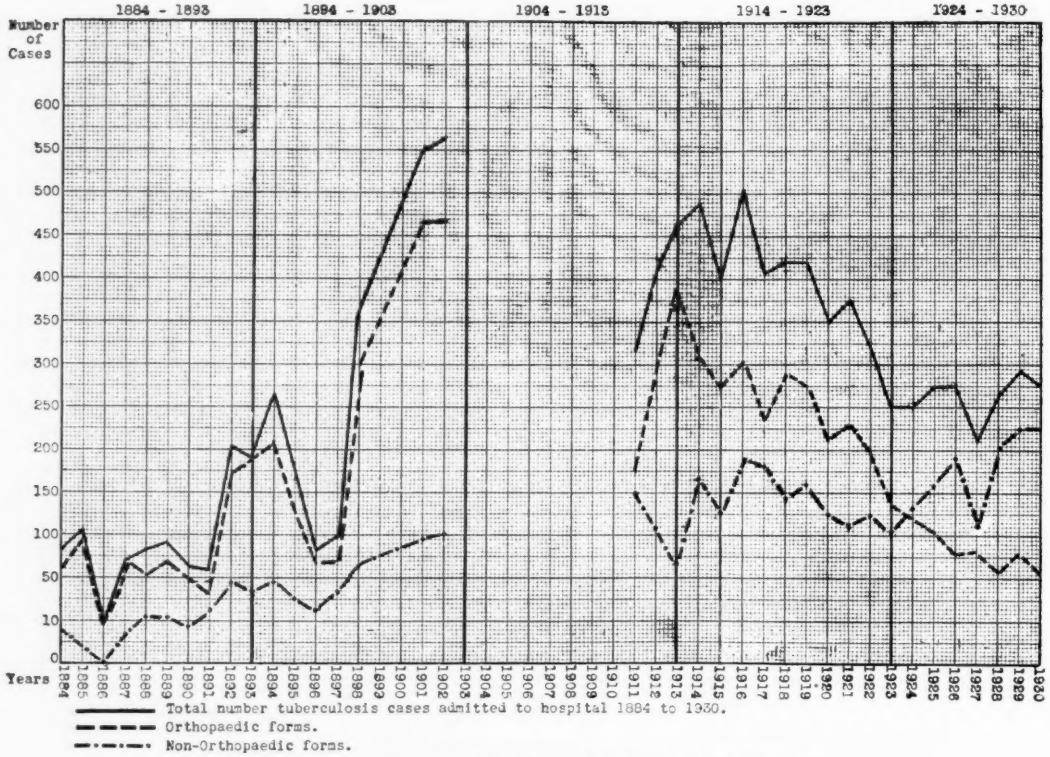

CHART I. Total number of cases of tuberculosis admitted to the Children's Hospital of Boston 1884 to 1930. Relation of orthopaedic to non-orthopaedic forms.

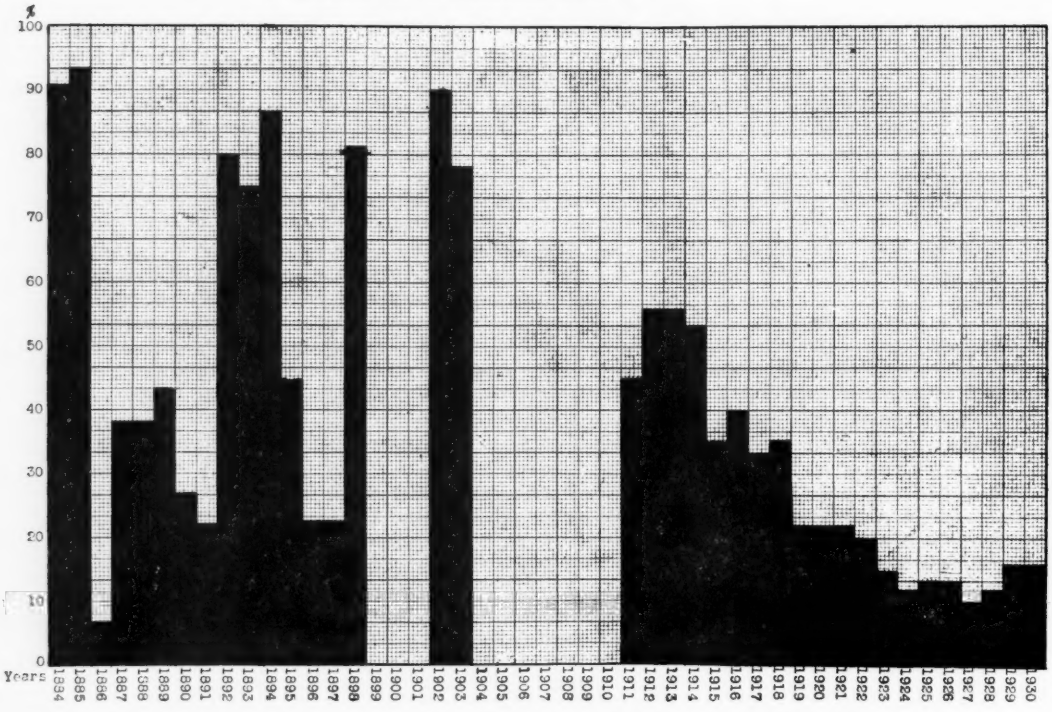

CHART II. Incidence of tuberculosis during years 1884 to 1930.

It is to be greatly regretted that data regarding tuberculosis cases are not available for the years 1903 to 1910. At this time, due to a change in the hospital's statistical method, cases were not classified by diagnosis.

Whether the curve would have gone up still higher or fallen during this period of nine years must be a matter of conjecture. However, a glance at the figures showing the death rate per 100,000 population from tuberculosis in Bos-

ton* leads one to believe that the decline in the number of cases of tuberculosis was gradual during these years reaching the comparatively lower figure in 1911.

For the following five years, 1912 to 1916 inclusive, the cases again increased in number but did not reach the height of 1902.

Beginning in 1917, there was a gradual decline in the total number of cases of tuberculosis admitted to the Children's Hospital. This continued for ten years until 1927. In 1928 there was a moderate increase which continued through 1929, followed by a very slightly lower number of cases in 1930.

During the forty-six years covered by this review the Children's Hospital admissions have increased from 914 cases in the year 1884 to 15,789 cases in the year 1930. In terms of the total admissions to the hospital, therefore, the percentage of cases diagnosed as tuberculosis was relatively higher in the early years, 1884 to 1902.

The highest incidence of tuberculosis occurred in the years 1884 and 1885 (9% and 10% respectively). For the subsequent six years the incidence was less, ranging from 0.7% in 1886 through 3.8% in 1887 and 1888 to 4.2% in 1889, dropping again to 2.7% and 2.1% in 1890 and 1891. In 1892 there was a rise to 8% remaining high for three years, 1892 to 1894 inclusive. In 1895 the percentage of cases was lower 4.5%, remaining low 2.3% for the following two years, 1896 and 1897. In 1898 it again rose to 8.1% reaching 9% in 1901, dropping to 7.8% in 1902. The figures for 1899 and 1900 were not available.

In 1911 the incidence was lowered to 4.5%, rising to 5.6% in 1912 to 1913. From then on a steady decline was noted reaching the lowest figure in 1927 (1.0%).

It is interesting to note that the death rate per 100,000 population from tuberculosis in Boston during the years 1917 to 1924 shows a drop in rate from 191.5 in 1918 to 100.8 in 1924†. A similar decrease in the mortality rate per 100,000 population from tuberculosis in Massachusetts‡ is noted for the subsequent years 1924 to 1928. In 1924 the rate was 86; in 1928 it was 73, a percentage decrease of 15. This is comparable to the lowered incidence of tuberculosis cases in the Children's Hospital during these years.

A further study of the statistics of these cases of tuberculosis shows that the orthopaedic forms were numerically greater than the non-orthopaedic forms during the first twenty years (1884 to 1903). The number of orthopaedic cases paralleled closely the figures for the total number of cases of tuberculosis so strikingly demonstrated in Chart I. The percentage of orthopaedic forms during this time, 1884 to 1903, varied from its highest in 1886 (100%) to the lowest in 1897 (70%).

During this period (1884 to 1903) the non-orthopaedic forms of tuberculosis were few in number and the percentage during this time was correspondingly low, no higher than 19% which occurred during the year 1888. From

TABLE II

Year	Total Admissions	Number of Tuberculosis Cases	Percentage of Tuberculosis Cases
1884	914	83	.09
1885	1,086	102	.093
1886	1,261	10	.007
1887	1,565	61	.038
1888	1,657	63	.038
1889	1,883	80	.042
1890	2,154	59	.027
1891	2,393	58	.021
1892	2,595	208	.08
1893	2,652	199	.075
1894	3,035	263	.086
1895	3,513	161	.045
1896	3,724	86	.023
1897	4,055	94	.023
1898	4,495	367	.081
1899	5,085	.....	.....
1900	6,180	.....	.....
1901	6,116	554	.09
1902	7,055	556	.078
1903	7,684	.....	.....
1904	7,896	.....	.....
1905	8,602	.....	.....
1906	8,212	.....	.....
1907	7,358	.....	.....
1908	8,612	.....	.....
1909	7,631	.....	.....
1910	7,910	.....	.....
1911	7,395	335	.045
1912	7,298	413	.056
1913	7,874	458	.056
1914	9,361	495	.052
1915	11,516	410	.035
1916	12,552	505	.040
1917	12,529	428	.034
1918	12,059	439	.035
1919	15,140	430	.021
1920	16,078	351	.021
1921	17,057	360	.021
1922	16,499	333	.020
1923	16,522	257	.015
1924	19,390	262	.013
1925	18,992	273	.014
1926	18,308	274	.014
1927	19,647	216	.010
1928	19,425	267	.013
1929	19,342	294	.015
1930	15,789	278	.015

*Tuberculosis Survey of Boston by M. P. Horwood, Ph.D.: "Death Rates Per 100,000 Population from Tuberculosis in Boston."

1903—236.4	1906—221.2	1909—189.3
1904—236.8	1907—206.0	1910—204.7
1905—230.4	1908—204.8	1911—185.2

†Tuberculosis Survey of Boston by M. P. Horwood, Ph.D.: "Death Rates Per 100,000 Population from Tuberculosis in Boston."

‡Whitney, J. S.: "Facts and Figures About Tuberculosis." National Tuberculosis Association, 1930.

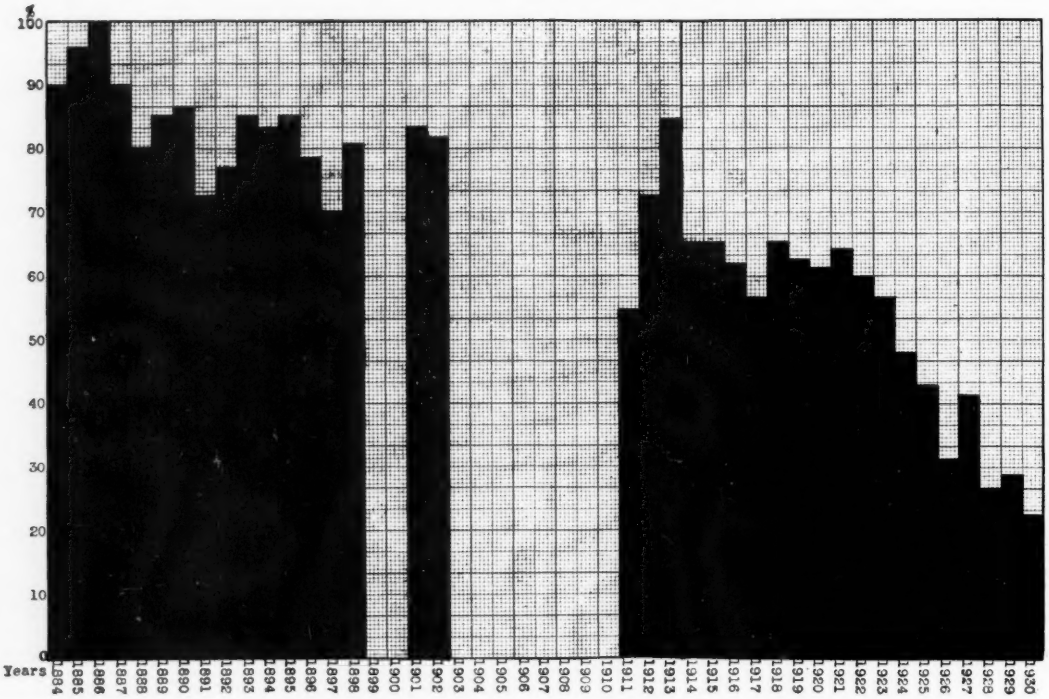

CHART III. Incidence of orthopaedic forms in cases of tuberculosis during years 1884 to 1930.

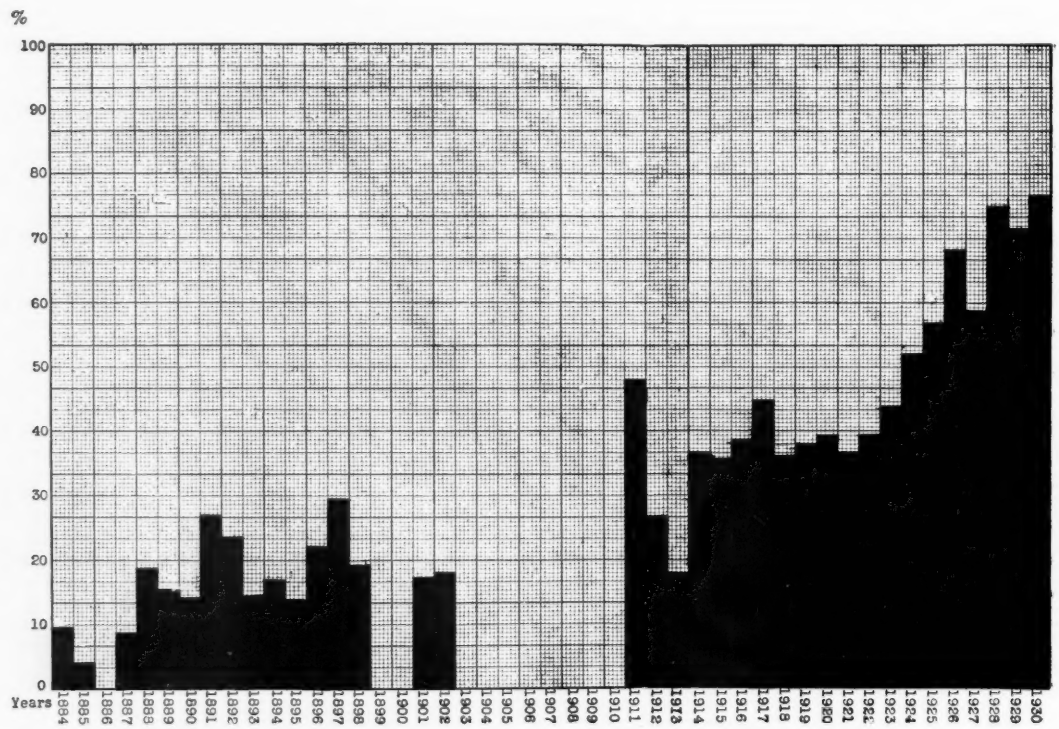

CHART IV. Incidence of non-orthopaedic forms of tuberculosis 1884 to 1930.

TABLE III

Year	Number of Tuberculosis Cases	Number of Orthopaedic Cases	Percentage of Orthopaedic Cases	Number of Non-Orthopaedic Cases	Percentage of Non-Orthopaedic Cases
1884	83	75	90	8	9.6
1885	102	98	96	4	3.9
1886	10	10	100	0	.....
1887	61	55	90	6	9.9
1888	63	51	81	12	19.0
1889	80	68	85	12	15.0
1890	59	51	86	8	13.7
1891	58	42	72	16	27.5
1892	208	161	77	47	22.5
1893	199	172	86	27	13.15
1894	263	217	83	46	17.4
1895	161	138	86	23	14.2
1896	86	67	78	19	22.0
1897	94	66	70	28	29.0
1898	367	298	81	69	18.8
1899	.....	.....	.....	.....	.....
1900	.....	.....	.....	.....	.....
1901	554	459	83	95	17.1
1902	556	457	82	99	17.7
1903	.....	.....	.....	.....	.....
1904	.....	.....	.....	.....	.....
1905	.....	.....	.....	.....	.....
1906	.....	.....	.....	.....	.....
1907	.....	.....	.....	.....	.....
1908	.....	.....	.....	.....	.....
1909	.....	.....	.....	.....	.....
1910	.....	.....	.....	.....	.....
1911	335	179	53	156	46.5
1912	418	303	72	115	27.5
1913	458	385	84	73	15.9
1914	495	326	66	169	34.1
1915	410	272	66	138	33.6
1916	505	313	62	192	38.0
1917	428	242	57	186	43.4
1918	439	290	66	149	33.9
1919	430	273	63	157	36.5
1920	351	215	61	136	38.7
1921	360	231	64	129	35.8
1922	333	199	60	134	39.1
1923	257	145	56	112	43.5
1924	262	127	48	135	51.5
1925	273	116	42	157	57.5
1926	274	85	31	189	67.9
1927	216	88	41	128	59.2
1928	267	66	25	201	75.2
1929	297	86	29	208	70.7
1930	278	61	22	217	77.7

1891 to 1903 there was a general increase in the number of non-orthopaedic forms, the highest incidence occurring in the years 1891 (27%) and 1897 (29%).

In 1911 the comparative figures of the orthopaedic and non-orthopaedic forms of tuberculosis show a spectacular reduction of the orthopaedic forms (53%) and a rise of the non-orthopaedic forms (46.5%). For two years after this, 1912 and 1913, the orthopaedic forms increased to 84%, while the non-orthopaedic forms decreased to 15.9%. From 1914 on there was a general lowering both numerically and in the percentage of orthopaedic forms of tuberculosis, the non-orthopaedic forms continuing a gradual increase both in number and percentage.

Probably the most interesting story told by the statistical study occurs from the years 1924 to 1930 (Charts I and II). During this period both the number and the percentage of orthopaedic forms show a steady decline, reaching the lowest point in 1930 (22%), while during these years the non-orthopaedic forms steadily increased to reach a percentage of 77.7 in 1930. This reversal of position of orthopaedic and non-orthopaedic forms of tuberculosis since 1924 leads to the thought that the practice of preventive medicine is more successful in the orthopaedic forms of tuberculosis.

SUMMARY

1. A statistical study of the cases of tuberculosis admitted to the Children's Hospital of Boston during the period of forty-six years, 1884 to 1930 inclusive.

2. A comparative study of the incidence of orthopaedic and non-orthopaedic forms of tuberculosis.

3. Figures demonstrate the high incidence of tuberculosis during the first 20 years covered by the study (1884 to 1903) compared with the diminished incidence in the last twenty years (1911 to 1930).

4. The incidence of orthopaedic forms of tuberculosis shows a high level from 1884 to 1903; a gradual lowering of the incidence from 1911 to 1924; a marked decrease from 1924 to 1930, reaching the lowest point (22%) of the total number of tuberculosis cases in 1930.

5. The incidence of non-orthopaedic forms of tuberculosis shows exactly the reverse of that of the orthopaedic forms. In the early years of the study, 1884 to 1903, the incidence of non-orthopaedic forms is low, reaching the highest point at 27% of the tuberculosis cases in 1891. In the last twenty years of the study, 1911 to 1930, the incidence rises rapidly reaching the highest level at 77.7% in 1930.

CONCLUSION

This study of the tuberculosis cases at the Children's Hospital of Boston has confirmed the impression that tuberculosis of the osseous system is a disappearing disease and that this form of tuberculois responds more favorably than the non-orthopaedic forms to the measures of preventive medicine.

THE GREATER BOSTON HOSPITALIZATION PLAN

AT a meeting of hospital representatives to consider plans for prepaid hospitalization charges held in Sprague Hall, of the Boston Medical Library, June 8, 1933, Dr. John W. Bartol, President of the Boston Health League, was unanimously elected chairman. There were nineteen representatives of hospitals of Greater Boston present.

In accepting the chairmanship Dr. Bartol stated that this plan had not been presented to the Executive Committee of the Health League and that the League was merely acting as the organization to furnish opportunity for discussion of this plan by the hospital group. Miss Tracy, Executive Secretary of the League, acted as secretary of the meeting.

The following draft was presented.

A Proposed Plan For Prepaid Hospitalization To Be Operated By Certain Hospitals Of Boston Through A Non-Profit Corporation To Be Known As The Greater Boston Hospitalization Association, Inc.

SUBJECTS TREATED

1. Brief Outline of the Plan
2. Beneficiaries of the Plan
3. Agreements between Association and Hospital Members
4. Agreements between Association and Subscriber
5. Plan of Management
6. Functioning Committees
7. Addenda:
   - Actuarial Soundness
   - Advantages to Hospitals
   - Management of Operation
   - Agreement Forms

GREATER BOSTON HOSPITALIZATION PLAN

The undersigned hospitals agree that it is of great importance that a step be taken to spread the cost of sickness amongst people of moderate means by a system of periodic payments of small sums by many in order to meet the large expenses of sickness amongst a few of these many. The present method or lack of method is happy-go-lucky, the individual gambles that illness will not strike him or his family, well knowing that if he loses the stake, it means the loss of his savings and very likely that he must go into debt to pay doctor and hospital or he must accept charity.

If present methods continue, with more and more patients being treated free and more and more hospitals driven to seek support from government, it is apparent that we are proceeding in the direction of "state medicine". It will soon be demanded that physicians and surgeons be on salary from the government and that hospitals be owned and controlled by government.

The layman, the prospective patient, has the right to plan and adopt a method of financing himself in time of sickness. He cannot dictate the amount that the hospital or the physician shall charge. If, however, certain hospitals agree to accept certain sums for their bills and if certain hospital staffs agree to accept definite sums for the payment of their bills at hospitals for patients from among these groups of people of moderate or small means, it is clearly his right to organize with others to meet these payments in a way that he can finance.

Illness which needs hospitalization is the most expensive illness, therefore a plan to cover this expense is the most urgent. A plan to meet the hospital bill alone would help the hospital which although an institution of fundamental importance to the community is having more and more difficulty in financing itself by the old methods of voluntary donations and charges to patients. It would help the doctors somewhat, because with the hospital bill out of the way his bill would be an earlier charge upon the patients' savings; it would help the patient somewhat because when the financial strain comes the hospital bill is cared for and he has as an unusual bill that of the physician—even so this bill may take all his savings and throw him into debt.

A plan whereby these groups of people of small means by periodic payments of small sums may meet the expense of hospitalized sickness by paying both hospital and physician's bill would be most helpful to the patient and would bring payment to the physician from many patients whom he now must treat free. It would put an end to free treatment of patients who ought to pay and who can pay by this method.

The plan would in no way change the existing relations between doctors and patients. Hospitals with closed staffs would not change their policy in this particular. There are, however, reputable hospitals which are open to the practice of physicians and surgeons not upon the staffs of closed hospitals. There are hospitals, more especially in the suburbs, which have large courtesy staffs. It is felt that the hospital needs of the family physician are adequately met in Greater Boston. The proposed plan would make no change in such relations. Hospitals may well do more in the way of helping physicians not on their staff to keep in touch with the advancements in medicine. This

can and should be done by the establishment of clinics open to the practitioners of medicine. Perhaps certain hospitals may also establish clinical laboratories for the examination of specimens and performance of diagnostic procedures possible only in laboratories connected with our large hospitals. This would operate to facilitate and improve the work of the general practitioner. More diagnostic clinics may well be established.

Plan A:

If the physicians on the staffs of the hospitals will agree to a scheme whereby their bills for these groups of hospital patients may be met as well as the hospital bills, that would be by far the best for all these, patients, hospitals and physicians. To do this a fee table must be agreed upon. In this community this may prove fairly simple because such a system is now in operation at the Baker Memorial of the Massachusetts General Hospital. This is conducted with the approval of the professional staff and was indeed first suggested by them.

Plan B:

If the staffs of the hospitals forming this Association are not yet prepared to agree to a scheme whereby payment of their bills to hospital patients may be included, then the hospitals will proceed with a more limited plan for the payment of hospital bills only.

Beneficiaries of the Plan

Individuals of employed groups, such as school teachers, employees of banks, utilities, department stores, commercial and industrial organizations.

It is recognized that it is very desirable to include the families of these individuals upon some additional payment. It is left for the Trustees to determine whether this shall be done at first and what the amount of this payment shall be.

The employee pays each month

Plan A—$2.00
Plan B— .90

which he authorizes his employer to deduct from his wages and remit to the Greater Boston Hospitalization Association.

A "certificate of membership" is issued by the Greater Boston Hospitalization Association to the employee in which a stipulated hospital service is to be rendered, in case of need, by any hospital that is a member of the Association.

The Greater Boston Hospitalization Association will pay to any hospital member $6.00 for each day's service rendered to subscribing members to a maximum of 21 days to each patient in any one year.

Members are secured only from selected employed groups considered good or fair risks, in order to preserve actuarial soundness.

The approach to the employee is through the employer. The coöperation of the employer is essential in approaching his employees and in operating the wage-deduction plan.

Agreements Between the Association and Hospital Members

Approved hospitals who become members of the Association enter into an agreement to render services to employee members who require hospitalization on the basis of terms stipulated in the following agreement which the Association makes with its subscribing member.

The Association agrees to compensate the hospital member rendering such service at the rate of $6.00 per day.

A tentative outline of the agreement made between the Association and its insured member follows:

Agreement Entered into Between the Greater Boston Hospitalization Association and the Wage Earner

In consideration of (Plan A) twenty-four dollars ($24.00) a year, or (Plan B) ten dollars and eighty cents ($10.80) a year, payable annually, semi-annually, or quarterly, or (Plan A) two dollars ($2.00) per month or (Plan B) ninety cents ($.90) per month, through voluntary payroll deductions, each payable in advance, the subscriber is entitled to participate in the benefits of the Greater Boston Hospitalization Association Plan on the terms set forth below.

1. The benefits hereinafter set forth are available to the subscriber immediately in the case of accident or emergency illness, and after 15 days from date of this contract in all other cases covered hereby.

2. Any hospital with which the Greater Boston Hospitalization Association has a contract for such services in force at the time hospital service is applied for hereunder may be selected by the subscriber.

3. The subscriber shall receive hospital care in a room designed to serve two or more patients. If the subscriber prefers a single room, a credit of $4.00 per day will be given on the price of the room selected.

4. Hospital care includes bed and board, general nursing care, routine laboratory examinations, emergency and non-ambulatory x-rays, operating room, routine medications and dressings, general anesthesia when such service is supplied by the hospital, and all other customary routine treatment which may be prescribed by the physician during the period of hospitalization.

5. Such hospital care and service will be furnished up to and including 21 days in any contract year, on one or more admissions, provided the aggregate days shall not exceed 21. (Additional service will be furnished to the sub-

scriber at one-third discount off the regular hospital charges.)

6. Plan A: The benefits hereunder include the services of a physician or surgeon who is chosen by the subscriber and who agrees to care for him, and who is authorized to practice in the hospital in question. The services of special nurses or their board are not included.

Plan B: The benefits hereunder do not include services of the subscriber's attending physicians, surgeons, special nurses or their board.

7. During the period of hospitalization the subscriber must be under the treatment and care of a physician in accordance with his staff privilege at the hospital selected by the subscriber.

8. The services hereunder will end at the time the subscriber is discharged as a hospital patient by his personal physician, and the subscriber will be responsible to the hospital for payment of its regular charges for hospitalization after the date of such discharge.

9. All service furnished hereunder is subject to the rules and regulations of the hospital selected by the subscriber. The benefits included hereunder cover only the treatment and care of illness and injuries regularly accepted for treatment by the hospital selected by the subscriber, and do not include cases provided for by Workmen's Compensation, pulmonary tuberculosis, mental disorder, quarantinable disease, drug addiction, alcoholism, or a chronic condition requiring a "rest cure", except in such hospitals as may care to take the particular case in question. Obstetrical cases will only be taken at the expiration of nine months' payment.

In case of an epidemic, public disaster, or other conditions making it impossible to provide accommodations to all eligible subscribers, the responsibility to provide hospital service would be discharged by the refund to the subscriber in need of hospitalization of the amount that had been paid by him on his twelve-months' contract in effect at that time. If services had been secured from one of the participating hospitals under the contract during the year of his membership, only a proportionate amount of the fee paid would be refunded.

No rights whatsoever shall accrue hereunder to any subscriber except for and during the period, whether annual, semi-annual, quarterly or monthly, for which the subscription fee has actually been accepted.

It would be expected that a Reserve Fund will be established. After a sufficient Reserve Fund is obtained lower membership rates may be given or provision made to include members of employees' families at a reduced rate, or some other form of benefit to subscribers adopted.

PLAN OF MANAGEMENT

1. Trustees:

Elected by General Council, composed of eminent citizens, philanthropically inclined.

2. General Council:

The General Council will be composed of the President or a member of the Board of Trustees designated for the purpose, the Chief of Staff or a member of the Staff designated by that body for this purpose, and the Director (Superintendent) of each member hospital. In addition there shall serve as members of the General Council two members of each County Medical Society in the district served appointed by such Society for this purpose.

NOTE: The bills of any hospital approved by the Association will be paid. That does not make such a hospital a member. Member hospitals are those which unite to form the Association and such others as they may elect. Affiliated hospitals are those not members approved for the payment of bills by the Association.

3. Functioning committees as follows:

Finance and accounting
Medical profession
Actuarial
Membership and promotion
Hospitals
Public Relations
Legal.

4. Approach to an employer through committee-men representing different types of business classifications.

5. The plan for securing membership hospitalization in the individual employed group unit.

6. General Summary.

FUNCTIONING COMMITTEES

Finances and Accounting:

This committee will supervise the system of accounting, make audits, approve expenditures, make reports and will have full supervision of all financial affairs of the Association.

Medical Profession:

This committee to be composed of a group of hospital staff doctors who will advise on any procedure involving the relation of medical staffs or the medical profession.

Actuarial:

This committee shall study actuarial findings as experience develops and make recommendations, if any changes are deemed necessary to contribute to soundness of operation. It would be well if an experienced actuary could be secured as a chairman of this committee. The

work of this committee will be of great value in determining the advisability of including or rejecting any particular group of employees as good or bad risks.

Membership and Promotion:

The duty of this committee will be to assist in securing the personnel for the various committees of classified business groups whose function will be to interest and secure the coöperation of employers in their respective groups.

It will call together from time to time the group chairmen of the classified groups for counsel and planning.

Hospitals:

The function of this committee will be to deal with any problems that arise involving the relation of hospitals generally to the Association.

If any question should arise as to the encouragement of malingering by any hospital in the group, such hospital's record will be reported to this committee for survey and recommendations.

They will also be consulted as to the advisability of including additional hospitals in the membership of the Association.

Public Relations:

The function of this committee will be to assist the Director in a proper presentation of the work of the Association in its relation to employers, employees and the public.

It might well include in its program of education and publicity a wider scoped presentation to the public of the work of the hospitals and their problems, thus developing better understanding and goodwill.

Legal:

To this committee will be referred all forms, agreements and other instruments used in the promotion of its work, as well as all other matters requiring legal counsel.

ADDENDA

Actuarial Soundness

Many factors enter into the operation of the Group Hospitalization Plan to ensure its actuarial soundness. Some of these factors are age limits, group risks, proportion accepted of men and women, percentage secured from each group, stability of employment, the wage deduction plan, and the scope of the agreement covering the services to be rendered by the hospital.

Maintaining a service agreement closely following the general terms offered by the one suggested on previous page, and using reasonable discretion in regard to other factors mentioned above, it has been found that under average conditions the following experience will apply.

Seven per cent of the members enlisted (over 1000) will require hospital care with an average stay of ten days. Thus, each one thousand members will require seven hundred hospital days' service. The above experience will be affected by any serious deviation in the terms of agreement for services to be rendered or modifications of the conditions under which memberships are received.

Other Advantages to Hospitals in Addition to Increased and More Widely Distributed Income

1. Increases bed occupancy with consequent reduction of per diem cost.
2. Avoids investigations and collection costs as well as unpaid accounts.
3. Creates goodwill among people of low incomes when bill is provided for in advance. Ill-will is sometimes created when patients are questioned or must be urged to pay part or all of bill.
4. Contributing public which must, either through tax or contributions, cover the deficit, will approve the effort to provide against such increasing burdens especially when, as at the present, incomes generally contract.
5. Enables voluntary hospitals to maintain their rightful place of leadership against unethical and unsound commercial efforts, attempting to capitalize on the growing demand for some provision for prepaid hospitalization.

Other Advantages to Physicians

1. Raises to the status of semi-private patients by whom through the Association the physician's bill is paid for services rendered in an approved hospital many patients who now receive free treatment from physicians.
2. Certainty of payment from many others who now accept service from the physician and find themselves unable to pay the bill.
3. Elimination of the unpleasant job of collecting from delinquents.
4. Maintenance of pleasanter relations with patients because of the elimination of these financial dealings.

The Board of Trustees

As in every corporation the Board of Trustees will be the responsible directing agency of the plan. It will have full authority in all questions of policy, management and operation. It will have the usual officers of secretary, treasurer, finance and executive committees, etc. It will employ its managing director who will be responsible to such Board as an executive officer.

Such a Board will give confidence and dignity to the movement in the minds of the em-

ployer, the employee and the public and will insure the permanency and solidarity of the plan. Membership to the Board of Trustees will be elected by members of the General Council described as follows:

The Board shall have the power to establish rates, determine upon the groups to be approached, hold custody of the funds, and authorize and make payments therefrom. The Board shall have broad powers. It may modify the plan as herein proposed but must carry out its general intent.

The General Council

The General Council will be composed of the President or a member of the Board of Trustees designated for the purpose, the Chief of Staff or a member of the Staff designated by that body for this purpose and the Director (Superintendent) of each member hospital. In addition there shall serve as Members of the General Council two members of each County Medical Society in the district served appointed by such Society for this purpose.

(1) From members of the Council appointments will be made by the Board of Trustees to the various committees outlined on preceding page.

(2) The Council will suggest policies and programs in the interest of the furtherance of the Association's objectives and refer them to the Board of Trustees for final action.

(3) Vote in proportion to number of beds available for this group of patients (semi-private). Each County Medical Society represented to have two votes.

It may be deemed advisable to include in the membership of the Council the Chairmen of the various committees of the business classified groups which will be referred to later.

The Approach to the Employer

The approach to the employer is probably the most crucial step in the operation of Group Hospitalization. The question of receiving his consideration will be very largely determined by two things. In addition to the benefits to be realized, he will want to know who is back of this movement and he will be very much affected by the person who approaches him on the subject. It is because of these two vitally important considerations in the approach to the employer that emphasis has been placed first, on a non-profit type of corporation managed by outstanding public men, and secondly, on the formation of committees representing the various classified groups of industry for the approach to employers within their respective groups.

In previous efforts in the large cities, where contacts have been necessary with employers in various civic movements, such as Chambers of Commerce, etc., business interests have been divided into 60 or more classifications.

One can readily see the strength that would come to this movement with the securing of prominent men representing each classified group, who with a committee of his selection, would act as sponsors in approaching employers. It is not difficult to compare the relative positions of a salesman coldly approaching an employer, as against that of an employed representative of the committee following up the lead of approach made through a member of his committee.

Presentation to Employer

In endeavoring to solicit the coöperation of the employer, we should emphasize the following:

(1) A plan whereby his particular business institution can put in operation hospitalization for his employees similar to other welfare plans undertaken by corporations for the last quarter of a century.

(2) Indicate how this can be done with a minimum of effort through clearing all administrative problems through the Greater Boston Hospitalization Association.

(3) Offer him the assistance of a Director in effecting the plan in his plant. Suggest the organization of a plant committee composed of foremen and other officers of his organization. Outline an approach to his employees including literature, speakers, etc. Include as members of plant committee the employment director and the one having charge of payroll records, or pay clerk.

Where a business concern operates as a plant unit, with the coöperation of its own local committees there is certain to be greater permanency and stability. The employment director can be constantly on the lookout for new members as additional employees are taken on.

Factors That Have a Direct Bearing On Per Cent Hospitalized and Length of Stay

1. Groups of employees are carefully selected on their risk ratings.
2. Wage earning employees hospitalized are naturally eager to resume employment.
3. Compensation cases involving serious injuries and frequently calling for longer stays are excluded in the plan.
4. Malingering—The administration plans provide for a continuous actuarial study covering employed groupings, individual hospitals, and even doctors. Thus study

would reveal the need of any correctives that might be found necessary. However, experience indicates a fine coöperative spirit, making correctives unnecessary.

SUMMARY OF ADVANTAGES OF MANAGEMENT PLAN

1. A non-profit corporation governed by a Board of Trustees composed of outstanding citizens, adding prestige and confidence to the plan, and assuring maximum acceptance by employers.
2. Complete control by the hospitals through the electing power of members to the above Board vested in the general council, which council includes in its membership the President, Superintendent, and heads of Staff of the participating hospitals.
3. The functioning of committees which deal with every important phase of the Association in its relation to the hospitals, staff, and medical profession, employers, employees and general public.
4. The coöperation in approaching employers by sixty chairmen and committees, who represent various classified business groups.
5. Operation in each business unit on a plan whereby its own entity is emphasized, thus securing maximum coöperation in the approach to employees and establishing continued interest and stability.
6. In the above plan the Association's entity is emphasized and the managing director, whose responsibility is not lessened, and whose effectiveness is many times increased in his relationship, takes his rightful place as an employed executive for the Association.

   This arrangement eliminates all possibility of the charge of commercialism, and avoids any procedure harmful to the best interests of the Association, member hospitals, and employed members.
7. Operation of such a plan will reduce administrative costs and allow a greater distribution of earnings to hospitals and make possible later on a reduction of fees or extension of hospital service to members.
8. Only on some such plan can the movement enter its field on a basis sufficiently broadened in scope to promptly and effectively meet the needs of the great numbers employed and at the same time become a real factor in broadening and strengthening the financial income of each hospital in the plan.

Suggested Agreement for Member Hospitals Between The Greater Boston Hospitalization Association and Hospital Members.

The Board of Trustees of the ..... Hospital at its meeting held .....day of ..... 19..... took action accepting membership in the Greater Boston Hospitalization Association of Boston, Massachusetts, and agrees to render service to subscribing members of said Association in accordance with the terms in Membership Agreement made between said Association and subscribing member, for which service the hospital will be remunerated by this Association at the rate of $6.00 per day or such sum as may be determined by said Association from time to time.

The said Hospital agrees to conform with the constitution and by-laws and other rulings that may be determined by the Association.

The Hospital agrees to give at least six months written notice of any decision to withdraw from the Association.

The following officers will represent the above Hospital on the General Council of said Association.

.....
President.

.....
Superintendent.

.....
Representative of Medical Staff.

.....
Name of Hospital.

.....
Secretary, Board of Trustees.

Suggested Agreement for Affiliated Hospitals Between the Greater Boston Hospitalization Association, Inc., and Hospitals Approved for Payment of Bills of Subscribing Members of the Association.

The Board of Trustees of the ..... Hospital at its meeting held .....day of ..... 19..... took action accepting membership in the Greater Boston Hospitalization Association, Inc., of Boston, Massachusetts, and agrees to render service to subscribing members of said Association in accordance with the terms in Membership Agreement made between said Association and subscribing member, for which service the hospital will be remunerated by the Association at the rate of $6.00 per day or such sum as may be determined by said Association from time to time.

The said Hospital agrees to conform with the constitution and by-laws and other rulings that may be determined by the Association.

The Hospital agrees to give at least six months' written notice of any decision to withdraw from the Association.

.....
Name of Hospital.

.....
Secretary, Board of Trustees.

Membership Application

.....193.....

I CERTIFY that I am in good health, ... years of age, married—single, and of my own knowledge have no chronic disorders or phys-

ical ailments, and hereby make application for membership in The Greater Boston Hospitalization Association, Inc., of Boston, Massachusetts, to become effective upon acceptance by said Association.

I authorize my employer to pay in advance for me $2.00 monthly (Plan A), or 90c monthly (Plan B), or $24.00 annually (Plan A), $10.80 annually (Plan B) by payroll deduction.

It is also expressly agreed and understood that default in the payment of any installment for a period of fifteen (15) days automatically cancels the contract, but if still in good health, I may at any time during a period of thirty (30) days after date of maturity pay the delinquent installments and a re-instatement fee of fifty cents (50c) and will then be entitled to the full benefits of the contract.

I am employed by .....

Name of family physician .....

You will advise me within ten (10) days if application has been accepted or rejected. If rejected, money is to be returned to me promptly; if accepted, I am to receive membership certificate.

False certification with reference to applicant's health will be basis for immediate cancellation of membership.

This application is made a part of the contract and is the basis on which the certificate is issued.

Phone, Business..... Name.....

Phone, Residence ..... Street Address.....

City ..... State.....

Checks Payable to .....

Treasurer

Greater Boston Hospitalization Association, Inc.
Boston, Massachusetts

Not-Transferable No. .....

Membership Certificate

THIS CERTIFIES THAT ..... is a member of the Group Hospitalization Plan of the Greater Boston Hospitalization Association, Inc., and upon payment to the Treasurer of said Association the sum of 90 cents monthly or $10.80 annually (Plan B) or $2.00 monthly or $24.00 annually (Plan A), in advance by the said party whose name appears above, said Association agrees to give said member hospitalization service at any hospital that is a member of this Association or approved by this Association, according to the following terms and conditions.

(Terms when approved to be inserted as part of this agreement.)

Failure to pay a periodic payment within fifteen (15) days from date due makes this contract null and void and of no further force and effect, except that upon satisfactory evidence of good health and upon payment of the unpaid amount within thirty (30) days from the due date of the last previous payment, plus a renewal fee of fifty cents (50c) the holder of this contract may, at the option of the hospital, be restored to full enjoyment of the benefits provided by this contract.

It is expressly agreed that neither said Association nor any of its agents or representatives have any authority to bind said hospital by any character of agreement in any way at variance with this written contract.

This certificate is for the period of one year from its date, and issued on the basis of conditions as set forth in the application signed by the member whose name appears above.

GREATER BOSTON HOSPITALIZATION ASSOCIATION, INC.

.....

President.

.....

Managing Director.

Dated ..... 193 .

Collection Agreement with Employers

The firm whose signature appears below agrees to make monthly payroll deductions of $2.00 each month (Plan A) 90 cents each month (Plan B) from all employees subscribing as members in the Greater Boston Hospitalization Association, Inc. of Boston, Massachusetts, on the written request of such subscribing employees, and remit same within five days of such deductions to the Treasurer of said Association at .....

.....

Name of Employer.

.....

Signature.

Deduction to be made each month on

.....

Date

DISCUSSION

MR. ROTCH, Boston Dispensary: A remarkable job has been done. If Plan A should be carried out in full I am afraid that the scheme would be set back on account of the money it entails. A comparatively small number of persons are going to come in on the basis of $2 monthly.

DR. WASHBURN: The figure $2 is arbitrary. The Trustees of the Greater Boston Hospital Association might find the figure might be set at loss. I personally believe that it can be sufficient.

MR. COLE, Newton Hospital: Has the possibility of transferring the certificate to an immediate member of the family been considered?

DR. WASHBURN: It has been left to the Board of Trustees to determine finally details such as the amount of money to be paid, whether dependents will be included, what diseases to bar,

if any, etc. These details can be determined only after proper actuarial advice.

MR. ROTCH: It is to be left to each hospital to determine whether it prefers A or B.

QUESTION: Have the medical societies taken any action?

DR. WASHBURN: Suffolk has appointed a committee to study the question—but no report has been given out.

DR. MCKITTRICK, Suffolk District Medical Society: The Committee consists of Dr. Harry Goodall, Chairman, Dr. Frank Lahey, Dr. T. J. O'Brien, Dr. Richard M. Smith, Dr. Channing Frothingham and me. Speaking unofficially, the Committee feels there will be a reaction against the plan by the general physician at this time so there is no official report by the Committee from Suffolk. The Committee feels that, at this time, no concrete plan would be happily received by the profession as a whole.

DR. CUTLER, Peter Bent Brigham Hospital: At the Annual Meeting of the Massachusetts Medical Society just past, I found that the President and the Council were opposing periodic payment plans because they had been misinformed. Had Dr. Stetson delivered his address on Monday his reference to the matter would have been entirely different. When Dr. Stetson learned that any hospital might enter into this plan he asked that a vote be passed by the Council and it is to be considered by them at an early meeting. In my opinion the success of the plan depends on every hospital in every community participating. Ninety per cent or more of physicians have hospital affiliations and if every hospital is included, one of the doctors' chief arguments in opposition breaks down.

DR. MCKITTRICK: I now speak as an individual. Much opposition at present is based on misunderstanding. A certain amount of education among the medical profession as a whole seems to me to be necessary. If a plan goes through now it will be without the coöperation of all the profession.

MR. BATCHELDER, President, Boston Lying-in Hospital: Plan B states payments are to be deducted from payroll. In the Emergency Campaign I was strongly against payroll deductions and my division raised one million two hundred thousand dollars. I believe payroll deductions will arouse opposition. Personally I am much interested and would like to see Plan A put into operation.

DR. WASHBURN: The plan is to be voluntary, as indicated in the outline.

DR. HOWLAND, Peter Bent Brigham Hospital: A plan is working in Houston and Dallas. I have written to Baylor University and they stress the necessity of enrolling large groups through employers. Success lies in large industrial groups as risks are then spread.

DR. CHRISTIAN, Peter Bent Brigham Hospital: Plan A considers the doctor. Physicians with few exceptions should oppose Plan B, which does not guarantee payment of their fee. If Plan A is adopted by the hospitals and presented to the physicians so that they realize that individually their payments are guaranteed, you are then in a position to ask their support of the plan. Two dollars monthly is much more than 90c but I feel that Plan B is doomed to fail because the physician has been left out. A large percentage of physicians have hospital privileges and therefore are directly interested in having their bills guaranteed. If we go to the physicians without this guarantee we are in a weak position. In the last analysis, the physician gives the greatest contribution to the philanthropy of the hospital by working without receiving a fee. Hospitals can no longer expect such service from physicians.

QUESTION: Who is going to pay this general manager and get the plan started? Is it necessary to have it underwritten? How much money do we need to start?

DR. WASHBURN: If hospitals would be willing to accept part payment, $3 instead of $6 a day at the beginning, the balance of the bill to be paid at the end of the first year, if there was sufficient surplus, hospitals could probably finance it. It has been intimated that some interested citizens would be willing to help inaugurate the plan. We can work out details after it has been decided to begin the plan. The plan has already been discussed. Why wait? I do not see why we cannot come to a conclusion now. If 19 hospitals take it back to their Trustees, let each hospital decide whether it wishes to come into the plan and whether it shall be A or B. If 10 hospitals are coming in, why not start? This is Greater Boston, so why wait for the Council of the State Medical Society to act? Suffolk has already been given the plan. If hospital staffs decide to participate, the medical societies will follow. There are three essential elements in the plan: Public, Hospital, Physician. I think it is good for all three or I would not advocate it.

FATHER BRENNAN, St. Elizabeth's Hospital: I feel there is an element in human nature which should be considered. In a metropolitan area such as this there is always a group that expects free hospital care. My experience in collecting bills leads me to believe that the man who has a steady job will pay his bill and on a small income he would be disinclined to make a monthly payment for future illness.

DR. ZINSSER, Massachusetts General Hospital and Harvard School of Public Health: From

a public health viewpoint, I am much interested in this suggestion. I do not feel that many persons are dead beats. It must be recognized that physicians and hospitals are giving service often on a charity basis and this is rapidly approaching the end. Free cases will have to be taken care of by taxation, so that hospitals and physicians are properly paid. This plan is devised for meeting emergencies that are not ordinary routine charges. Whether physicians like it or not, due to the general attitude of the public, there are plans now for medical insurance with control by an insurance company which undertakes the risk. Details of the plan here depend on activities to be undertaken and the size of the group. Final details must be worked out after the plan is started. Medical insurance is going on—particularly in foreign countries—and the difficulty in many of these countries is that the medical profession does not manage the plan, and there is as a result state medicine. The ideal plan would be one initiated and managed by the medical profession, thereby permitting the profession to maintain control of its own affairs. Since such a plan is impossible, the best substitute seems to me to be this plan which is to be initiated by the hospital with the approval of its staff and trustees.

MR. WHITMAN, Massachusetts Memorial Hospitals: Is this plan insurance or not?

DR. WASHBURN: The legal committee of the Massachusetts General Hospital is of the opinion that it does not come under the insurance laws, but is a contract.

QUESTION: Is a corporation to be formed, or a loose association of individuals?

DR. WASHBURN: Hospitals might work it out either way. If it is worked out under the insurance law, more money must be raised.

NEW ENGLAND BAPTIST HOSPITAL: Would each participant be required to have a physical examination?

DR. WASHBURN: If the group is sufficiently large it can be done without examination as the spread is sufficiently great.

DR. COOK, New England Deaconess Hospital: Has scheme A been tried anywhere?

DR. WASHBURN: Only Plan B—in about 20 places. Hospital associations have sponsored Plan B because of medical opposition to Plan A.

MISS LADD, Faulkner Hospital: Our hospital is in a community where the people have moderate means, incomes from $1,000 to $5,000 yearly. It is extremely difficult for this group to pay for hospital illness without exhausting their life savings. Personally, I feel something should be done to help them meet the cost of illness. This plan will make it easier for them. As to the attitude of our staff, already six or eight members have asked if it would not be possible to inaugurate a plan similar to Baker Memorial for Faulkner.

MR. BOWDITCH, Faulkner Hospital: I wish to corroborate Miss Ladd's statement. The majority of persons coming to Faulkner do wish to pay their bills and often they find them a burden which it takes a long time to pay.

DR. CUTLER, Peter Bent Brigham Hospital: I move that this body give general approval to Plan A and that a copy be sent to each hospital in Greater Boston and to each county medical society within this district asking that they give the plan study and that they send representatives to a subsequent meeting.

FLOOR: Favor general motion but omit specific mention of Plan A.

MR. PRESTON, Massachusetts Memorial Hospitals: At the present time in my opinion physicians' opinions should be obtained.

REPRESENTATIVE OF THE NEW ENGLAND BAPTIST HOSPITAL: Physicians feel that their functions would be interfered with.

DR. BREED, Massachusetts General Hospital: It is futile to discuss whether physicians approve until after the hospitals have given approval. This meeting, which is unofficial, is in favor of approving the general scheme as set forth in the plan with tentative approval of Plan A.

MR. FITZPATRICK, Trustee, Peter Bent Brigham Hospital: Is it wise to begin now without study of group insurance? During the depression many companies have been obliged to give up group insurance and many companies have abandoned complete and partial disability clauses.

DR. POLLOCK, Massachusetts Memorial Hospitals: This group cannot commit itself to any definite plan. After each hospital has considered the matter, call a second meeting to discuss details.

CHAIRMAN: There is a motion before the meeting.

QUESTION: Should these minutes and this plan be sent to all hospitals?

DR. WASHBURN: This scheme involves member hospitals and affiliated hospitals, and that membership be limited to the larger hospitals. There will be many hospitals, where the bills will be paid, who are not members and the council by vote can add any hospital to the original

group, but do not make the original organization too unwieldy. Dr. Cutler has asked that the hospitals represented in this group take the plan back to their trustees and staff and decide whether they wish to come in and has moved that a letter be sent to all other hospitals in Greater Boston asking that they study the plan and decide whether they wish to participate. This will make the group unwieldy.

DR. CUTLER: My object in including all hospitals is to educate the physicians. If the staffs of all hospitals were asked to study and discuss the plan and reach a decision regarding it, I think we shall be in a much stronger position. I, therefore, amend my motion to read

"I move that this body give general approval to the principle of the plan and that a copy of the plan be sent to each county medical society and to every reputable hospital in Greater Boston, asking their trustees and staff to give careful consideration to it, and in addition ask the hospitals represented here today to become the association."

DR. HURLEY, Carney Hospital: I wish to state that I view the plan with interest.

DR. BARTOL: The motion which is still before the meeting calls for additional work which seems to entail some slight expenditures of money—Is anyone prepared to assume this responsibility?

DR. WASHBURN: I will.

DR. CUTLER: I move that my motion be amended to include a time limit and that replies to the letter be sent to Dr. Bartol by July 1.

DR. BARTOL: I will repeat Dr. Cutler's motion:

Moved: that this body give general approval to the principle of the plan and that a copy of the plan be sent to the county medical societies and to every reputable hospital in Greater Boston asking their trustees and staff to give careful consideration to it. In addition the hospitals represented here today are asked to form the association and are further asked that they reply to Dr. Bartol before July 1 regarding their interest in the plan.

The motion was carried.

MR. WHITMAN: I move that a copy of the plan be sent to the Insurance Commissioner requesting that he decide whether it be insurance.

DR. CHRISTIAN: I feel the plan should not go to the Commissioner until a definite plan has been acepted by the hospitals.

DR. BARTOL: All in favor of sending a copy to the Insurance Commissioner please signify by aye—opposed by nay. The motion is lost.

The Chairman then declared the meeting adjourned. MARGARET H. TRACY, *Secretary.*

A BILL TO SUPPLANT THE PRESENT FEDERAL FOOD AND DRUGS ACT

Declaring that nearly 27 years' experience in the enforcement of the national pure food and drugs law has disclosed many serious deficiencies in its provisions, the Secretary of Agriculture, Henry A. Wallace, by direction of President Roosevelt, June, 1933, submitted a bill designed to supplant the present Federal food and drugs act.

Seven of the outstanding and most significant provisions of the proposed new food and drugs law are briefly outlined in the Secretary's letters to the Committee Chairmen.

(1) The prevention of false advertising. The need for the control of serious abuses in this field is generally recognized.

(2) The inclusion of cosmetics. Serious injuries have occurred through the sale and use of cosmetic products of a character harmful to the user. The practice of deceptive labeling and advertising of such products should be regulated.

(3) The establishment of tolerances for added poisons in foods. A complete elimination of all poisonous substances from foods may be impossible. Where the presence of poisons is unavoidable, their amounts should be kept so low that by no possibility will the food be harmful to health. The present law contains no provision authorizing the establishment of tolerances for poisons, but imposes upon the Government the obligation of showing affirmatively in every case that a food contafning poisonous substances may be harmful to health independent of similar adulterations prevailing extensively in other foods.

(4) Authority for the Secretary, after notice and hearing, to promulgate definitions and standards for foods which will have the force and effect of law. Under the present law such authority extends to the field of canned foods only. Legal food standards like those now existing for official drug products are essential for efficient enforcement operations.

(5) Power to require manufacturers to procure in certain cases Federal permits. This method of regulation will be invoked in those instances only where a menace to health is involved and where adequate protection to the public cannot be effected through the other provisions of the bill.

(6) The control of drug products on the basis of therapeutic claims which are contrary to the general agreement of medical opinion. This removes the burden imposed by the present law of proving not only that such statements are false but that they were made with fraudulent intent on the part of the manufacturer. This has seriously handicapped the Department in the extension of adequate protection to the public in the purchase of drugs labeled as treatments for various diseases.

(7) Requirement of informative labels. With one or two minor exceptions the present food and drugs act contains no positive labeling requirement. It merely prohibits false or misleading statements and these prohibitions are to some extent minimized by excepting provisos. A disclosure on the label of sufficient facts to enable intelligent and discriminating buying will operate unquestionably to the advantage of the consumer.—*U. S. Dept. of Agriculture.*

CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE MORTEM AND POST MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

EDITED BY RICHARD C. CABOT, M.D.
F. M. PAINTER, A.B., ASSISTANT EDITOR

CASE 19281
SUDDEN UNEXPECTED DEATH OF A PHYSICIAN

PRESENTATION OF CASE

DR. BURTON E. HAMILTON: This is a case of sudden death in a sixty-two year old male physician.

Family history. His mother lived to be seventy-one and died of angina pectoris. His father died a senile death associated with pneumonia at seventy. One brother died after several years of disability from senile heart disease at seventy-six. One sister aged sixty-seven is living. Two sisters died in childhood, one of typhoid. Although the senile changes in this family do not appear until about seventy, it seems to me that in comparison with contemporary families of the same stock, the symptoms of old age have appeared in this patient's family perhaps ten years earlier than usual.

The past history is essentially unenlightening. He was suspected of tuberculosis in late adolescence and took a year off. During this year he changed from a tall, thin boy to a robust, heavy man. He played football, tennis, squash and golf and continued his athletic habits throughout his life. He took frequent satisfactory vacations. He had sufficient sleep. He controlled his weight. He had no bad habits; he took no alcohol and used tobacco after his forty-fifth year only. He was, however, throughout his life under continuous emotional strain. It is hard to exaggerate the emotional excitement under which he lived. He took his patients' problems hard, and became elated over their successes as much as he suffered over their disasters. In spite of his emotional personality he never showed neuroses or psychoses.

He had reported to me for physical examinations at irregular intervals for ten years before his death. The examinations were entirely negative. His blood pressure had never been recorded as above 130/85. Electrocardiograms, the last taken six months before his death, were entirely normal. Roentgenological examination showed no changes in the heart and great vessels. On one occasion only, several years ago, urinalysis showed a yellow reaction to the sugar test, but blood sugar tests were normal.

On the day of his death he worked and took some moderate exercise during the morning, ate a large luncheon, came back to his office fatigued, told his secretary that it was deplorable that a man of his age could not rest after luncheon but that he had no time that day. He made one professional call, returned to his office to pick up some medical supplies on his way to a hospital, but did not leave the office. He telephoned my home, talked with a member of my household and left word for me that he had a pain under his sternum, but that it was not severe and that if I was busy not to hurry but to come when I could and that he would wait in his office for me. He then chatted apparently comfortably for a while. After he had rung off, the message was given to me. I went immediately to his office. On reaching there, not more than ten minutes after his telephone call, I found him lying on his office table unconscious, breathing rapidly, deeply, stertorously, and somewhat dusky in the face. My first impression was that it was a cerebral accident. I tore his collar open; there was nothing interfering with his breathing. I felt his pulse, which was present and not rapid or slow. As I examined him his breathing became irregular and his right arm and leg stiffened. Others came at once from their offices, and we were able to give him artificial respiration, to try to start the heart by direct pressure on the chest wall and below the ribs and rather promptly to inject adrenalin into the region of the heart, and directly over the heart with a long needle.

CLINICAL DISCUSSION

DR. HAMILTON: There is not much comment. He had never complained of anything suggesting angina before, but of course the substernal pain which he mentioned over the telephone forces this diagnosis. Common as sudden deaths from angina are, we do not actually witness the deaths of many of our patients, and in spite of the fact that his only known symptom was angina, his appearance when unconscious suggested so strongly a cerebral lesion that I asked Dr. Mallory to include the brain in his examination.

DR. PAUL D. WHITE: How long was the time interval between his call for help and your finding him in coma?

DR. HAMILTON: There was an interval of about ten minutes between his telephone call and his death. He appeared to have nothing wrong when he left his office and he came back about an hour later. We do not know when his pain began. At the time he telephoned he was not in severe distress.

DR. TRACY B. MALLORY: This is a type of case which is of course not uncommon but one which we do not have a chance to discuss in this hospital very often. This type of patient does not get to the hospital before death. I am very grateful to Dr. Hamilton for being willing to come down and present the history.

DR. WHITE: Most of the patients whom I

have known who have died of angina pectoris have died very suddenly. I am not sure about the exact interval between the onset of pain and death. In two or three instances that I recall very well it was a matter of seconds, in others it was apparently instantaneous. Patients who have coronary thrombosis may possibly die as suddenly as those with angina pectoris due to the fulminating effects of the pain or some other mechanism. Those that survive may live any length of time. In this instance there may have been coronary occlusion with death not instantaneous but after a period of twenty minutes or more, or there may have been angina pectoris with collapse following it, that is the so-called syncope angiosa, which condition has long been on record. The patients whom I have seen who have fainted with angina pectoris have recovered at least for the time being; they have not died in coma. Whether he would fit into that group as a rare exception or show an occluding blood clot in the coronary circulation, which clot might be hard to find, I do not know.

DR. J. H. MEANS: I should like to ask about cyanosis in death from angina pectoris. Did you say he was cyanotic?

DR. HAMILTON: Not deeply. He impressed me as rather red in the face and breathing hard. The appearance was in contrast to a death in angina I witnessed two days ago when apparently the heart beat ceased, then the breathing stopped and the patient became intensely cyanotic, stiffened and died within a minute; this suggested a Stokes-Adams attack from cessation of the heart beat. In the present case, there was cyanosis rather than pallor, but the cyanosis was not intense.

DR. WHITE: Most patients with severe coronary pain have pallor rather than cyanosis during the acute attacks.

DR. MEANS: My impression is that in a severe attack of angina one observes pallor rather than cyanosis.

DR. HAMILTON: I think the main thing is that there are general vasomotor disturbances associated with angina. They vary in degree. There may be an appearance of pallor or of cyanosis. There may be faintness up to loss of consciousness.

DR. MEANS: The reason I brought the point up is that I thought of a patient you autopsied here some time ago who had a sudden seizure, became unconscious and deeply cyanotic, and at autopsy showed narrowing of the coronaries without any thrombosis. I wonder if that may not be the condition here. In that case we had known for some months that he had something wrong with his heart. He had a gradually falling blood pressure with increasing tachycardia. I shall be interested to know in the present case the width of the coronaries as well as whether they contained thrombi or not.

CLINICAL DIAGNOSES

Angina pectoris.
Coronary thrombosis?

ANATOMIC DIAGNOSES

Coronary sclerosis.
Arteriosclerosis.

PATHOLOGIC DISCUSSION

DR. MALLORY: The autopsy was negative except for the heart. We examined the brain and found nothing there whatever.

The heart was not hypertrophied, weighing 350 grams, which I should say is quite normal for a man as large as he was. All branches of both coronary arteries were markedly narrowed but nowhere thrombosed. I examined them by multiple cross sections and they were either quite empty or contained fluid blood. I will show you the extent of the microscopic changes.

The transverse branch of the left coronary artery shows numerous atheromatous deposits. There is marked narrowing and in some places almost complete disappearance of the muscle cells in the media. There is a great deal of fibrous thickening of the intima but no proliferation of the elastica. The lumen is reduced to about one third or one quarter the normal size. The descending branch on the left has an extremely small lumen, again I should estimate as only a quarter of the normal diameter. In some areas the degree of narrowing is even more marked.

This is a fat stain and shows at this level narrowing down to one sixth of normal as the result of atheromatous deposits. Though there is very little persisting lumen, there is no suggestion whatever of thrombosis. One thing that interested me is the very marked active fatty degeneration in the media as well as atheromatous deposits in the intima. There is still one other point. In this one section there is a little calcification in the intima in one spot, though in general the process has not gone further than one of fatty deposits.

DR. WHITE: There is one other mechanism that should be mentioned as a possibility and that is abnormal heart rhythm. Ventricular fibrillation should be mentioned as a possible cause of death. But here was a man who was lying unconscious with an apparently normal heart rhythm. The pulse was regular when Dr. Hamilton took it, so that an abnormal rhythm would be unlikely as the cause of death. Until we can take electrocardiograms during death in angina pectoris we cannot be perfectly sure of the mechanism. It is my belief that the heart suddenly stands still in such cases.

A STUDENT: Is there any way that you get any estimation of a situation such as that before death? In this man there was apparently no information.

DR. HAMILTON: No, I do not think there was

in this case. In coronary disease there are clinically three groups; one in which the symptoms appear as part of a generalized arteriosclerosis, the angina being one of many complications; another group in which the first evidence one has of any defect is angina or coronary occlusion; a fairly large group have no evidence of disease until the first or fatal attack of angina or coronary occlusion. We are prepared to advise the first two groups. It would be very satisfactory if we could by health examinations discover evidence to anticipate the diagnosis in the third group, but certainly in many cases, like this one, we have no adequate means. Perhaps we should be more careful in giving régimes to men over forty. Perhaps those of us who are over forty might even carry nitroglycerin with us as a possible aid in a possible emergency.

DR. WHITE: He would not have concealed his pain for five years?

DR. HAMILTON: No, I think not. I saw him nearly every day. He was not particularly concerned about angina, though we talked about it, as many physicians who are over forty do. It seems to me that he would have mentioned any symptoms suggesting angina at once, as he did when his fatal attack began.

DR. HOWARD B. SPRAGUE: I should like to ask if anyone here has any idea as to the relation between prolonged physical activity, athletics, and coronary changes.

DR. HAMILTON: My opinion is biased in favor of regular exercise and perhaps is not worth anything. This patient had kept in very good physical condition and there were no warning signs. I approved of his continued regular exercise. Last summer I looked up the immediate factors in death or disability with angina and (1) prolonged usual activity with fatigue and (2) unusual physical exertion appeared as the commonest immediate factors. I should be inclined on that basis to put down the immediate cause in this case as fatigue. He had been working fairly hard with his practice, exercising as usual on the day of his death, had eaten a large meal and returned to work without rest. He had no reason to believe that he should not do the things he had been doing. But he was evidently tired and expressed his sense of fatigue. If I had been more alert, I should have advised him to rest one hour after his meals. I realize this does not answer your question.

DR. MEANS: I think the point about his emotional strain is very important. In the first place I think athletics are sometimes injurious, at least the punishment kind. In the second place I think mental and nervous strain are even greater causes of cardiovascular decay. I heard a most interesting talk by Dr. William J. Mayo a few years ago about doctors popping off one after another with coronary thrombosis. He gave us quite a speech about the rôle of emotions in the practice of medicine. This man took his practice seriously and really did worry about it a good deal. If one could practice medicine without worrying one would be less likely to have sudden death from cardiac causes. I do not know how that can be brought about.

DR. WHITE: I would agree with Dr. Means that nervous strain is much more important than physical strain as a background of angina pectoris. The patients I have seen with angina pectoris may or may not be athletic, but in almost every instance there is serious nervous strain.

DR. MALLORY: I talked with Dr. George Burgess Magrath about this case. As I said before, in a hospital of this sort we see very few of these cases, whereas the medical examiners see, relatively speaking, a very considerable number of them, and agree that as a rule these cases show no actual thrombi in the coronary arteries but almost invariably show markedly diseased coronary arteries, as this man did.

DR. WHITE: The cases of angina pectoris that I have seen postmortem have all shown some involvement of the coronaries, contrary to some reports, except for one in which there was bacterial endocarditis and vegetations extending from the aortic valve to the mouths of the coronary arteries and a few cases of luetic aortitis with narrowing of the coronary mouths.

I once asked Dr. George Magrath if he had ever come across any cases of sudden death without adequate explanation at postmortem, presumably from spasm of the coronaries, that is without coronary sclerosis. He said he never had.

CASE 19282

HYPERTENSION, DIASTOLIC MURMUR AND PRECORDIAL BULGE WITH SUDDEN DEATH

MEDICAL DEPARTMENT

PRESENTATION OF CASE

DR. HENRY D. STEBBINS:* This is another case of sudden death. The patient was a forty-four year old Irish-American, unemployed. He complained of dyspnea and dizziness of eight months' duration and a choking sensation on the day of admission.

Nine months before entry he went to another hospital and had an operation for bilateral inguinal hernia. He was thoroughly examined and it was felt that there was nothing wrong with his general physical condition. There is no information as to his blood pressure at that time. Eight months before entry he noted a thumping sensation over his heart on exertion, with shortness of breath. He also noticed that when he got up, sat down or leaned over he suddenly became dizzy. His doctor informed him that his blood pressure was 185. At that

*Recently senior interne on the East Medical service.

time he had slight headaches. Seven months before entry he first noticed nocturia once or twice a night. Very frequently in the evening as he was sitting quietly in a chair he noticed a slight choking, oppressive sensation (not pain) in his chest running up to the left root of the neck. Four months before admission he got up suddenly in the middle of the night and fell down. He was very dizzy, more than in his previous attacks, but did not become unconscious. Following that attack he had an increase of dyspnea, but was not kept from his usual activity.

Two months before admission he came to the Outpatient Department, where he was seen by Dr. Breed. His blood pressure at that time was 210/116. His heart was 10.5 centimeters to the left of the midline, with the midclavicular line 8 centimeters. There was a definite precordial bulge. He had a long diastolic murmur at the aortic area but no Corrigan pulse or sign of aortic regurgitation. A seven-foot heart film showed the heart only slightly enlarged, with a widening or slight bulge of the aorta. His blood Hinton test at that time was negative.

The patient was not seen again until the day of admission to the wards, when, while sitting on the bench outside the Cardiac Clinic, he suddenly felt a severe choking sensation in his upper chest and felt as if he were going to die. He suddenly felt very weak and collapsed, but did not become unconscious. He had never had an attack of this sort before. He had had no reason to limit his activities before this time. He was seen by Dr. Palmer, who sent him to the Emergency Ward with a tentative diagnosis of cardiac asthma.

The past history was negative. There was no history of cardiovascular disease in the family. He denied venereal disease.

When seen in the Emergency Ward he was lying in bed more or less comfortably, not propped up very high. He was definitely but not markedly cyanotic, sweating slightly, but not very dyspneic. The pupils were slightly irregular but reacted well to light. There was marked bulging of the precordium to the left. The apex impulse of the heart was palpable 10 centimeters to the left of the midline, though not forceful or heaving as described in the Outpatient Department. The midclavicular line was 8 centimeters from the midline. The left border of dullness could be percussed 10.5 centimeters out in the fifth interspace. The supracardiac dullness was 5.5 centimeters. His heart sounds were of good quality. There was a long coarse systolic murmur at the base, loudest to the right of the sternum, about 2 or 3 centimeters out in the second interspace. This murmur was definitely transmitted to the right carotid and was louder there than at the base of the heart itself. It was never heard over the left carotid. The pulsation of the right carotid artery was more marked than that of the left as one saw it in the neck. There was a sharp blowing diastolic murmur over the same area as the systolic, but not transmitted to the vessels. The blood pressure was 210/100. No Duroziez's sign was elicited over the inguinals, and the radial pulse was regular and of fairly good quality, not collapsing in type. Otherwise the physical examination was essentially negative.

He was given a quarter grain of morphia with considerable relief immediately on arriving at the Emergency Ward. About two hours later on his way to the Medical Ward he suddenly became unconscious and began to have Cheyne-Stokes breathing. He did not show any sign of pain. On arrival in the Medical ward he was in collapse, deeply cyanotic, heart sounds very faint, rate 70 but regular, pulse not obtainable, blood pressure not obtainable. He was given nitroglycerin sublingually, then intravenous adrenalin and caffein with slight decrease in cyanosis and slightly greater regularity of his respiration but no very great change in the quality of the heart sounds. About five minutes afterward the heart sounds were almost inaudible and the breathing was very irregular. In spite of intracardiac adrenalin he died.

Clinical Discussion

Dr. Stebbins: At the time of the patient's first visit to the Outpatient Department it was felt that probably the best diagnosis with the information at hand was rheumatic heart disease with aortic regurgitation and secondary hypertension. This was considered more likely than hypertension with functional regurgitation or than syphilitic disease of the aorta because of the long duration suggested by the precordial prominence.

On the patient's second visit to the Outpatient Department, about a week later, Dr. Breed considered primary hypertension and arteriosclerotic heart disease with secondary regurgitation as the most likely diagnosis, because of the small heart and widened aorta.

The discharge diagnosis was very much in doubt. The possibility of a ruptured aneurysm was considered but discarded for lack of positive evidence, leaving the nature of the final episode uncertain. Aortic regurgitation with secondary hypertension probably due to old rheumatic heart disease was felt to be the most likely underlying cause.

Dr. Robert S. Palmer: Do you remember what his pulse was?

Dr. Stebbins: It was not rapid, 66.

Dr. Palmer: In the Outpatient Cardiac Clinic I thought two things were queer about him, the slow pulse, a normal rate in spite of his difficulties, and the fact that his chest was perfectly clear although he was very dyspneic.

Dr. Stebbins: I have the pulse noted here

as 72 when he arrived in the Medical ward, and I remember saying at that time that it was peculiar that his rate had not increased since I saw him in the Emergency Ward. This was his second collapse, therefore we definitely discredited the diagnosis of cardiac asthma.

DR. PAUL D. WHITE: Where was the pain?

DR. STEBBINS: He did not complain of pain.

DR. WHITE: What was the attack in the Cardiac Clinic?

DR. STEBBINS: A choking sensation extending up to the left root of the neck, with collapse, extreme weakness and dyspnea.

DR. WHITE: It did not go through to his back?

DR. STEBBINS: No.

MR. DANA FARNSWORTH*: I saw this patient and he told me that he had pain from the top of his head down to about the twelfth thoracic vertebra.

DR. WHITE: In addition to the pain in front?

MR. FARNSWORTH: Yes. I remember that very distinctly because I wrote up an account of the symptoms.

DR. GEORGE W. HOLMES: The heart shadow I think is enlarged. The left ventricle is the most prominent portion. It is rounded as though there were some real hypertrophy present. There is marked increase in the supracardiac shadow with a definite bulge in the ascending aorta and some prominence in the knob. It does not seem to be dilated. Here the arch dips over to the left. It seems normal, and in the oblique view so far as we can make out the aorta is of normal width. I do not see anything to suggest aneurysm of the thoracic aorta or of the innominate. We have had a few cases where the aneurysm was at the root of the aorta and to the left posteriorly that we have missed, particularly if it was a dissecting aneurysm.

It would be interesting to know the x-ray report, including the fluoroscopic findings.

DR. STEBBINS: The examination was made two months before he died. "The heart shadow is slightly enlarged to the left in the region of the ventricle. The curve of the left ventricle is blunt. There is also definite increase in the supracardiac shadow. There is a distinct bulge of the aorta to the right. In the oblique view the root of the aorta seems wide. The aortic knob however is not flattened. There is no evidence of dilatation in the transverse or descending aorta. Hypertrophy of the left side of the heart, dilatation of the ascending aorta".

DR. WILLIAM B. BREED: I think there are two things that should be explained. One is the bulging precordium and the other the pure diastolic murmur from the region of the aortic valve with no systolic murmur at all, which one might expect to find in the roughened aorta of arteriosclerotic origin or aortitis. That was the main reason why the first time I saw him I suspected pure aortic regurgitation of rheumatic origin.

*Fourth year student working in the Cardiac Clinic.

At the second visit I shifted from that diagnosis, chiefly on the basis of the history and the x-ray picture, which could not be explained entirely on the basis of rheumatic heart disease with aortic regurgitation.

Another interesting point is the development of a loud systolic murmur later on in the course of his episode just prior to death. The clinical diagnosis as far as we were concerned up to that time was hypertensive heart disease with a dilated aorta and a dilated aortic valve.

DR. TRACY B. MALLORY: Have you anything to add, Dr. Palmer?

DR. PALMER: Nothing, except that when I saw him he was a desperately sick man with difficulty in breathing and complaining of a pressure sensation over his chest. I thought he had cardiac asthma. Even though his pulse was normal I still thought it was cardiac asthma and I sent him to the Emergency Ward for morphine and venesection, which I thought would probably relieve it.

DR. WHITE: I agree with Dr. Breed and Dr. Palmer. There might or might not have been actual deformity of the aortic valve here with hypertension complicated by aortic regurgitation, and as for the cause of death, one naturally thinks of coronary thrombosis as the most probable cause. Was there any evidence of congestion in the lungs?

DR. STEBBINS: No, sir.

DR. WHITE: Oppressive pain in the chest was the presenting symptom in this acute attack rather than dyspnea, and if we would take much account of the pain in the back, we might be led towards the correct diagnosis; we encountered not long ago a somewhat similar case. Angina pectoris may radiate to the back, but such radiation is extremely unusual; angina pectoris is generally substernal and may radiate to arms or neck but almost never to the back. This pain was distinctly referred down the back, which fact might have suggested the diagnosis.

CLINICAL DIAGNOSES (FROM HOSPITAL RECORD)

Hypertension.
Hypertensive heart disease.
Aortic regurgitation.
Acute cardiac failure.
Lues?

ANATOMIC DIAGNOSES

Medionecrosis aortae cystica.
Rupture of dissecting aneurysm of the aorta into the pericardium.
Hemopericardium.
Cardiac hypertrophy, hypertensive type.
Benign vascular nephritis.
Pulmonary tuberculosis, healed, with calcification.
Cholesterosis of the gall bladder.

PATHOLOGIC DISCUSSION

DR. MALLORY: At autopsy I think we were all very much surprised. We found that the aorta was quite markedly dilated. As we started to

open it we cut through one layer, came down on some fluid blood and suddenly found that there was another apparently complete aorta within the first one. It was then obvious that we were dealing with a dissecting aneurysm which had dissected completely the entire length of the aorta, starting from a small rupture in the endocardium just above the aortic valve and running all the way down to the common iliac vessels. It had also dissected upward into the innominate and subclavian arteries and a small way up one carotid into the neck. Exactly how long the aneurysm had been present I think there is no way of telling. The blood in the newly formed vessel was not clotted. It was fluid everywhere. There was no definite endothelial lining. He might well have had this dissection for some period of time, because there are several cases on record where dissecting aneurysm has occurred, the patient has made complete recovery, and later at autopsy has been found to have two aortas, one inside the other.

The immediate cause of death was more readily explained and is the common final event in these cases, the rupture of the outer wall of the aneurysm into one of the serous cavities. In this case it was the pericardium. There was about 400 cubic centimeters of fresh blood in the pericardium, and tamponade was in all probability the mechanism of exitus. In the other cases of dissecting aneurysm we have seen there has been a very marked degree of arteriosclerosis. In this man there was almost none, and instead we found a perfect example of the so-called medionecrosis aortae idiopathica cystica which Erdheim[1, 2] described four years ago and which Moritz[3] has recently reported upon in this country. This can probably occur at any age and frequently is the precursor of dissecting aneurysms in relatively young persons in whom arteriosclerosis is quite out of the question.

Here is a microscopic slide of one of the cystic cavities in the middle of the media. They all run along at about the same level and it is apparently at the convergence of one of these cavities with another that eventually a large space is formed through which the pulsating blood dissects its way.

In the phosphotungstic stain one can see that in this middle zone of the media the muscle fibers are almost entirely wanting. Here is the inner layer of the media with the normal distribution of muscle, then a zone almost devoid of muscle again, and then the adventitia. There is almost complete destruction of muscle fibers in this middle zone.

With elastic tissue stain one can see that the elastic tissue in this middle zone has not disappeared as it would in various other types of aortitis. In fact the elastic fibers here appear blacker and thicker than elsewhere.

In connective tissue stains the collagen strands are still present in normal amounts. They show up as blue fibers throughout the section. In this stain the muscle, which is red, is absent, the collagen, which is blue, is present, so that there is a fairly specific disappearance of the muscle alone. Just for contrast, here is the typical luetic aortitis, showing the foci of complete destruction of muscle and elastic tissue in the aorta with the puckering and beading of the intima over the area of scar. It is a totally different picture.

DR. BREED: Were the coronaries entirely normal?

DR. MALLORY: Yes. The dissection had reached down around the mouth of the right coronary and there was some degree of narrowing of the vessel by pressure from without, but the intima of the arteries was practically negative.

DR. WHITE: Was there any evidence that the dissection might have occurred in stages?

DR. MALLORY: No, there was nothing anatomical to suggest that, though the final rupture of the outer wall into the pericardium I am sure occurred at a later date than the formation of the aneurysm itself.

DR. WHITE: Could you say whether it was hours, days or weeks later?

DR. MALLORY: I think the rupture of the pericardium, that final episode on the way to the ward, was a matter of a few minutes.

DR. WHITE: Can you explain the discomfort in the morning and for a half hour or two before death as being the beginning of the dissection?

DR. MALLORY: It may have been, but we should then find difficulty in explaining his symptoms of a month or so earlier. He had nothing but a mild hypertensive heart disease at that time so far as we can make out.

DR. WHITE: How heavy was the heart?

DR. MALLORY: The weight was estimated at 500 grams.

DR. WHITE: He had had some shortness of breath previous to his final acute illness, and this shortness of breath was very probably due to his hypertensive heart disease.

DR. HOLMES: Was the rupture of the aorta close to the aortic valve?

DR. MALLORY: Yes.

DR. HOLMES: It seems to me hardly possible that the condition found at autopsy could have been present when the films were taken. We got an excellent view of the innominate artery and of the arch of the aorta, and any dilatation would have shown. We have not a very good view of the ascending aorta, but it does not seem possible that he had had as much dilatation as that.

DR. WHITE: Clinically the history suggests that the dissection probably occurred that morning.

1 Erdheim, J., Medionecrosis aortae idiopathica. Virch. Arch. f. path. Anat. 1929, 273:454.
2 Erdheim, J., Medionecrosis aortae idiopathica cystica. Virch. Arch. f. path. Anat. 1930, 276:188.
3 Moritz, Alan R., Medionecrosis aortae idiopathica cystica. Am. J. Path. 1932, 8:717.

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THE TRIBUTE TO DR. ROBERT B. OSGOOD

THE present number of this *Journal* contains several articles which are contributions from the Orthopedic Department of the Children's Hospital. These contributions are the results of years of work and observation and represent for the most part original work on the part of the writers.

This number is dedicated to Dr. Robert B. Osgood as a compliment from the Staff of the Children's Hospital, as most of the work set down in the individual papers went on while he was chief of the Orthopedic Department and the John B. and Buckminster Brown Professor of Orthopedic Surgery, Harvard University.

Two of the contributors have died, Sir Robert Jones, Bart., who held Dr. Osgood in high esteem, and Robert Soutter, who was connected with the Children's Hospital for nearly thirty years.

It is hoped that this number of the *Journal* will be of interest to many of our readers and that it will also show in a way the type of work being carried on at the Children's Hospital. It does not represent everything that was accomplished during the term of Dr. Osgood's service (1922 to 1931), but is only a small cross-section of what is constantly being done.

THE GREATER BOSTON HOSPITALIZATION PLAN

QUESTIONS affecting medical service and economic features relating thereto have been raised in many sections of the country and plans for meeting the conditions in various localities have been put into operation. Some seem to have merit and others are looked upon with suspicion especially by the medical profession.

In Massachusetts many meetings have been devoted to discussions of suggested plans as well as to methods now existing. Several recommendations designed to deal with the problems of the laity, the hospitals and physicians were presented at a meeting in the Boston Medical Library, June 8, 1933. The records of that meeting appear on page 88 of this issue.

Two plans are presented which are designed to provide hospital care for groups of employees. In that designated as Plan A the arrangement in force at the Baker Memorial of the Massachusetts General Hospital, whereby the payments by the patients cover the charges of attending physicians and the hospital, is incorporated, but in the other, Plan B, this is omitted.

The underlying purpose of these plans is to induce groups of working people in good health to pay regularly designated amounts out of income into a common fund. Experience has shown that very many industrial workers do not make provisions for hospitalization and a very large proportion of the population is sooner or later confronted with illness which necessitates hospitalization without personal reserve or insurance.

These plans are forms of insurance but are designed to carry the average cost without profit to the managing organization other than the actual expense of administration. The parties especially in interest under these plans include the laity, the hospitals and the doctors. Another important group consists of the supporting hospital donors who have been essential factors in the development and maintenance of hospitals. It is obvious that this support will be less in the immediate future and all reasonable effort should be made to induce the laity to meet the expense of their illnesses. A large proportion gambles on health, is driven to indigence, and becomes a burden on hospitals, doctors and communities.

In a general way it may be stated that everybody should be concerned with respect to the necessity of adequate support of hospitals because they are indispensable agencies in dealing with the severer forms of illness. Without them

progress in medicine would be seriously handicapped because their facilities for study, training of practitioners and research are great contributing factors in dealing with disease. Their importance is beyond question and they must be maintained.

The interests of the medical profession, in the great variety of coördinated efforts designed to overcome defects in dealing with illness, are vital and must be considered. Hospitalization or individual attention is absolutely dependent on physicians and disaster will result if any scheme ignores the doctor, but on the other hand practitioners cannot be indifferent or unreasonably obstructive in the presence of an awakened determination to discover defects of medical service and find solutions.

In the present instance efforts are being made in Boston to bring about possible changes for the better as shown in the two plans which have been submitted. The reaction in the profession seems to be, to a degree, distrustful of the scope and details of both plans with especial doubt about Plan A as indicated in the fear that if adopted there would result diversion of patients from the family adviser because of the general impression that many doctors in Greater Boston do not have hospital affiliation. The facts are, as shown by a survey, that 71 per cent of doctors in this section have hospital affiliations, that 19 per cent are engaged in laboratories, teaching services or positions which are not concerned with family practice. This leaves only ten per cent who would be thus affected, a minority so small that it does not seem to weigh heavily in favor of this argument. A part of this plan is to provide technical service to doctors even though not affiliated with hospitals and to permit attendance on clinical exercises. A careful reading of the plans and the discussions will be instructive.

There seems to be abundant reason to fear that unless the medical profession can be led to be constructively interested in suggested changes that other elements will enter into the study of the problems of medical care for the low income groups and exercise more or less undesirable control.

The menace of state medicine is more than an idle dream. It has invaded private practice because of obvious reasons. The effective defense reaction will depend upon the broadening of the doctor's conception of changes which are taking place, and more definite organization of the profession for concentrated study of conditions and possible remedial measures. The doctor has been too long an individualist interested in his personal problems; he must take cognizance of the accomplishments of aggregations of persons with a common interest. To this end we implore the profession to give careful study to any plans relating to the problems of medicine. Boston is trying to lead the way. The Suffolk District Medical Society should exhibit leadership in constructive effort to approve meritorious and discredit obnoxious movements in its immediate section.

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

JONES, SIR ROBERT*. K.B.E., C.B., F.R.C.S., F.A.C.S. At the time of his death he was Lt. Col. Royal Army Medical Corps. He wrote the Foreword to the Tribute to Dr. Robert B. Osgood. Page 51.

OBER, FRANK R. M.D. Tufts College Medical School 1905. Clinical Professor of Orthopedic Surgery, Harvard University. Assistant Dean in Charge of Courses for Graduates, Harvard University. Surgeon-in-Chief, Orthopedic Department, Children's Hospital, and New England Peabody Home for Crippled Children. Surgeon to Poliomyelitis Department, Vermont State Department of Health. His subject is "Tendon Transplantation in the Lower Extremity." Page 52. Address: 234 Marlboro Street, Boston.

SOUTTER, ROBERT*. A.B., M.D. Harvard University Medical School 1898. F.A.C.S. At the time of his death Dr. Soutter held the following positions: Surgeon, Children's Hospital, Long Island Hospital and Surgeon-in-Chief, Massachusetts Hospital School. Member of Orthopedic Surgery Teaching Staff, Harvard University Medical School. Fellow, Boston Surgical Society, Boston Orthopedic Society and several foreign scientific bodies. His subject is "A New Operation for Slipping Patella." Page 59.

LEGG, ARTHUR T. M.D. Harvard University Medical School 1900. F.A.C.S. Assistant Professor of Orthopedic Surgery, Harvard University Medical School. Associate Orthopedic Surgeon, Children's Hospital. His subject is "Tensor Fascia Femoris Transplantation in Cases of Weakened Gluteus Medius." Page 61. Address: 319 Longwood Avenue, Boston.

SEVER, JAMES WARREN. M.D. Harvard University Medical School 1901. F.A.C.S. Assistant Professor of Orthopedic Surgery, Harvard University Medical School. Associate Surgeon, Children's Hospital and Boston City Hospital. His subject is "Changes in the Epiphysis—Secondary to Infection." Page 62. Address: 321 Dartmouth Street, Boston.

FITZSIMMONS, HENRY J. A.B., M.D. Harvard University Medical School 1908. F.A.C.S. Instructor, Harvard Medical School. Assistant Surgeon, Children's Hospital. Consulting Orthopedic Surgeon, Quincy City Hospital; Choate

*Deceased.

Memorial Hospital, Woburn; Providence Hospital, Holyoke and St. Mary's Infant Asylum and Hospital, Dorchester. His subject is "Congenital Torticollis. Review of the Pathological Aspects." Page 66. Address: 520 Beacon Street, Boston.

BREWSTER, ALBERT HOWELL. B.A., M.D. Johns Hopkins University School of Medicine 1918. Instructor in Orthopedic Surgery, Harvard Medical School. Surgeon, Orthopedic Department, Children's Hospital. Member, American Orthopedic Society. His subject is "Countersinking the Astragalus in Paralytic Feet." Page 71. Address: 234 Marlborough Street, Boston.

FITCHET, S. M. B.A., B.P.H., M.D. Harvard University Medical School 1921. F.A.C.S. Assistant Surgeon, Children's Hospital and Outpatient Department, Massachusetts General Hospital. Consulting Surgeon, Massachusetts Eye and Ear Infirmary and Josiah B. Thomas Hospital. Associate Surgeon, New England Baptist Hospital. Assistant Orthopedist, Courses for Graduates, Harvard. His subject is "'Flexion Deformity' of the Hip and the Lateral Intramuscular Septum." Page 74. Address: 319 Longwood Avenue, Boston.

MORRIS, ROBERT H. M.D. University of Toronto Medical School 1922. Assistant Orthopedic Surgeon, Children's Hospital. Instructor in Orthopedics, Harvard Medical School. Orthopedic Surgeon, Long Island Hospital. Member of Board of Medical Advisers, Industrial School for Crippled Children. His subject is "Foot Stabilization. A Review of Fifty-Two Operations." Page 78. Address: 253 Newbury Street, Boston.

KATZEFF, MIRIAM G. M.D. Boston University School of Medicine 1925. Junior Assistant Surgeon, Orthopedic Department, Children's Hospital. Clinical Assistant, Orthopaedic Out-Patient Department, Cambridge Hospital. Orthopedist, New England Hospital for Women and Children. Her subject is "Tuberculosis in Infancy and Childhood. A Statistical Study." Page 83. Address: 321 Dartmouth Street, Boston.

MISCELLANY

MORTALITY RATES

Telegraphic returns from 85 cities with a total population of thirty-seven million for the week ending June 17, indicate a mortality rate of 10.6 as against a rate of 10.1 for the corresponding week of last year. The highest rate (18.1) appears for Tacoma, Wash., and the lowest (5.4) for Yonkers, N. Y. The highest infant mortality rate (13.9) appears for Hartford, Conn., and the lowest for Canton, Ohio, Duluth, Minn., Evansville, Ind., Fall River, Mass., Grand Rapids, Mich., Miami, Fla., Somerville, Mass., Spokane, Wash., Syracuse, N. Y., Tampa, Fla., and Waterbury, Conn., which reported no infant mortality.

The annual rate for 85 cities is 11.6 for the twenty-four weeks of 1933, as against a rate of 12.1 for the corresponding period of the previous year.—*Bureau of the Census.*

A RADIO MESSAGE PREPARED AND SPONSORED BY THE COMMITTEE ON PUBLIC EDUCATION OF THE MASSACHUSETTS MEDICAL SOCIETY FOR THE DEPARTMENT OF PUBLIC HEALTH

EAT THAT YOU MAY HAVE SOMETHING TO EAT WITH*

BY LEROY M. S. MINER, D.M.D., M.D.†

On the basis of scientific investigation, we can no longer get away from the fact that what we eat determines to a considerable extent what we have to eat with—the teeth. For this reason, members of the dental profession are laying more emphasis than ever before upon the supervision and balance of their patients' diets, in order to stem, if possible, the rising tide of dental disease. And since dental disease is so widespread, affecting more than 100 million people in the United States, it is well worthwhile to listen to what the modern dentist has to say about diet and its relation to health in the mouth and health in the body.

Regulation of the diet serves two purposes. First, it can do much to maintain strong teeth, free from disease, in the adult's mouth. Secondly, it determines largely the character and quality of children's teeth, thus becoming an agent for the actual prevention of disease. Dietary control, to be effective as a preventive measure, must go back before birth to the period of pregnancy. It must be continued faithfully throughout the years of growth and development—that is, up to 18 years. Supervision of the diet gives good assurance of strong healthy teeth in the growing youngster, and it is in the prevention of disease that we are most interested today.

From this standpoint the child's nutritional background has two aspects; before and after birth. Before birth, the expectant mother's diet largely determines the quality of the baby's first teeth, for the calcification or hardening of the temporary dentition begins in the fifth week of pregnancy and is practically completed when the baby is born. The baby's own diet during infancy and childhood determines largely the quality of his permanent teeth. The first permanent tooth, called the six-year-molar because it appears in the sixth year, starts to calcify at birth, and this process of calcification of the remaining teeth continues until all the permanent ones are in place at the age of eighteen or twenty years. During pregnancy and

*Broadcast April 25, 1933, 4:30 P.M., WBZ.
†Dean of the Harvard University Dental School.

afterward, both mother and child are naturally under the care of an obstetrician or pediatrician who exercises control over their diets and determines the details. But from the dentist's standpoint there are certain fundamental dietary needs of the body which must be met, if oral health is to be maintained in the mother or insured for the child.

To understand the essentials of a sound diet, it is first necessary to know something of the different types of foods and their particular uses in nourishing the body. These classes are as follows:

1. Proteins—for general growth, muscles, and the repair of tissues.
2. Carbohydrates and fats—for the body's activities or energies.
3. Iron—for good red blood.
4. Calcium and phosphorus — for firm, strongly built bones and teeth.
5. Other minerals and the vitamins—for health, growth and protection against disease.

The foods which come under these different classes are as follows:

1. For proteins—milk, cheese, eggs, meat, fish, peas and beans, nuts and the whole grain cereals.
2. For carbohydrates—milk, bread, cereals, sugars, fruit and vegetables. For fats—milk, cream, butter, cheese, oils, meat, fish and vegetable fats.
3. For iron—vegetables, dried fruits and berries, egg yolk, molasses, meat, liver and kidney.
4. For calcium and phosphorus — milk, cheese, vegetables, fruits, whole grain cereals, and eggs.
5. For vitamins—milk, eggs, fresh fruits and vegetables, butter and cod liver oil.

In listening to this list of foods under the different classes, perhaps you were impressed by the frequency with which certain foods, namely, milk, eggs, fruits, vegetables and meat are found throughout. These foods serve so many requirements of the growing body that they are often termed the many-sided foods.

Now the interesting fact about our modern civilized American diets is that while proteins, carbohydrates, fats and iron are generally found in adequate quantities for the body's needs, shortages or deficiencies to a greater or lesser degree in calcium, phosphorus and the vitamins are often discovered. From the dentist's standpoint, these substances are regarded as extremely important, and the foods containing them, notably milk, eggs, fresh fruits and vegetables, and meat, should never be neglected in planning the daily repast.

These foods are known as the protective foods. Scientific investigation has shown that the plentiful eating of these foods will do much toward stemming the rising tide of dental disease. Many children, in whom tooth decay is rampant, show a marked decrease in the number of cavities when their diets have been changed to include more milk, fresh fruits and vegetables and to lessen candy and sweets. Moreover, adults generally exhibit an improved condition of mouth health when their diets have been revised and balanced.

Now this whole question of diet has been the subject of a great deal of publicity during the last ten or a dozen years. It is interesting tc observe, probably as a result of this publicity, how the eating habits of the people have changed since before the war. People are becoming more conscious of the benefits to be derived from proper eating, and unquestionably much good has been done.

But publicity on dietary problems has also brought with it certain attendant evils. There has been a great tendency on the part of many well-intentioned but uninformed persons who take too literally what physicians, dentists and dietary experts have been quoted as saying. This has led to the development of fads in eating habits, fads that have often proved to be more injurious to the body than the original faulty eating. For example, many persons fired by a zeal to reduce weight, often when such reduction is not indicated from a health standpoint, have almost entirely eliminated carbohydrates and fats from their diets. Others have made their food intake largely vegetable with the almost complete elimination of meats in any form. Still others discuss seriously "incompatible" foods, and eat nothing but proteins for one meal, nothing but starches for the next, and so forth, on the theory that following such a régime will reduce them or failing to follow it will make them ill.

All these fads have little basis of scientific fact. They often fail to accomplish the desired end, and occasionally may themselves produce even more serious conditions. Probably the most important factor in this question of diet is *balance!* To achieve that balance from day to day requires only a small amount of intelligent thought, applied to make sure that a reasonable proportion among all the different foods is maintained and that no aspect is neglected. We can afford to be more liberal in our intake of milk, and this does not mean we have to drink it, for milk in cooked foods is also beneficial. We can afford to emphasize fruits for dessert rather than pastries and sweets. We can afford to use one or more green vegetables with each meal, especially if they are raw and fresh. Remember that raw cabbage in salad or other form is extremely valuable. Orange juice, eggs, whole wheat toast and butter are the fundamentals of a satisfactory breakfast.

But in following any set of directions such as these, common sense *must* be used. In the last analysis, the easiest way to solve the problem is to develop a taste for a wide variety of foods, and

then proceed to satisfy that taste in adequate fashion, without worrying from meal to meal as to whether enough Vitamin C is contained here, or too much carbohydrate is contained there. If you are ill, if you are much overweight or underweight, get a doctor's advice on your diet. Otherwise eat pretty much what you want, within reason, and don't worry about it.

COMPARISON OF DISEASE INCIDENCE IN CONNECTICUT WITH 1932 AND SEVEN YEAR AVERAGE

MONTH ENDING JUNE 24, 1933

Diseases	1933 Week ending June 3	1933 Week ending June 10	1933 Week ending June 17	1933 Week ending June 24	Average cases reported for week corresponding to June 24 for past seven years	1932 Week ending June 4	1932 Week ending June 11	1932 Week ending June 18	1932 Week ending June 25
Cerebrospinal Men.	—	—	—	—	—	—	—	2	—
Chicken Pox	172	184	128	110	62	120	170	80	66
Conjunctivitis Inf.	—	—	—	—	—	1	8	2	—
Diphtheria	—	2	4	3	15	3	4	—	4
Dysentery Amebic	—	—	—	—	—	—	1	—	—
Dysentery Bacillary	—	—	—	1	—	—	—	—	—
Encephalitis Epid.	1	—	—	—	—	—	—	—	—
German Measles	5	7	—	1	18	—	1	1	1
Influenza	2	1	3	—	—	2	1	—	—
Malaria	—	—	—	—	—	1	—	—	—
Measles	289	191	123	134	155	221	255	193	198
Mumps	97	79	38	66	28	71	69	57	29
Paratyphoid Fever	1	—	—	—	—	1	—	—	1
Pneumonia (Broncho)	33	9	13	17	14	21	22	17	8
Pneumonia (Lobar)	24	11	18	13	16	33	16	21	16
Poliomyelitis	—	—	1	1	1	2	—	3	3
Scarlet Fever	54	62	39	56	34	76	77	73	52
Septic Sore Throat	1	2	4	1	2	2	1	3	4
Tetanus	1	—	—	1	—	—	—	—	—
Trachoma	—	—	—	1	—	—	—	—	—
Trichinosis	—	—	—	—	—	—	1	—	1
Tuberculosis (Pul.)	30	31	29	32	36	27	43	35	40
Tuberculosis (O. F.)	2	1	2	—	4	2	6	3	3
Typhoid Fever	3	1	—	2	—	1	—	1	1
Undulant Fever	—	—	—	1	—	2	3	1	—
Whooping Cough	86	49	45	53	46	90	83	98	60
Gonorrhea	23	48	75	70	32	32	28	26	59
Syphilis	52	34	41	66	38	31	48	30	79

Remarks: No cases of Asiatic cholera, glanders, plague, or yellow fever during the past seven years.

CORRESPONDENCE

THE PHILANTHROPIES OF WILLIAM JOHNSON WALKER

May 29, 1933.

Editor, *New England Journal of Medicine*,

Thanks to the Boston Medical Library, referring to our meeting May 23, 1933 In Memoriam, William Johnson Walker, M.D. (1790-1865) I was able to exhibit Dr. Walker's one hundred page address of eighty-eight years ago (May 29, 1845) as printed (1848) Vol. VII, pp. 171, et seq. Portraits of Dr. Walker and of his father, Major Timothy Walker, were exhibited in the lecture room.

Dr. Walker, born in Medford, Mass., March 15, 1790, graduate of Harvard College, 1810, studied medicine in Medford, Paris, and London, and at a critical period of their history, gave approximately $1,000,000.00 to the following institutions, which were represented with speakers as follows:

Dr. Glover M. Allen

For the Boston Society of Natural History

Professor Henry G. Pearson
For Massachusetts Institute of Technology
Charles A. Andrews, College Treasurer
For Amherst College
Professor Halford L. Hoskins
For Tufts College
President Harry A. Garfield
For Williams College

All five speakers gave admirable addresses.

Dr. Walker, son of Timothy and Abigail (Johnson) Walker, was named for his grandfather William[5*] Johnson of Woburn (Burlington) of distinguished ancestry, being descended through William[4], Ebenezer[3], Major William[2], from Captain Edward[1] Johnson, of Canterbury, England, and Woburn (1637), author of the early history of New England styled "Wonder Working Providence." The date and place of Dr. Johnson's birth have been incorrectly printed. In his third year, on January 3, 1793, his father, Timothy Walker, "of Medford," by So. Middlesex Deeds, Lib. 112, fol. 32, bought his first Charlestown real estate of one Thomas Wood of Rutland for £100 and soon removed from Medford to Charlestown where William Johnson Walker had his early training in the public schools. After his graduation from Harvard College in 1810 he returned to Medford to study medicine and surgery under Dr. John Brooks (later for several years Governor of Massachusetts), but he appears to have received his degree of M.D. from the "Massachusetts Medical College," so-called, located on Mason Street, Boston. Afterwards he studied surgery under able surgeons in the Quartier Latin, Paris, and under Sir Astley Cooper in London. There is a tradition in the family that, at twenty-five, Dr. Walker served in a field hospital at the Battle of Waterloo in 1815. Dr. Walker's citations eighty-eight years ago clearly show his familiarity with the French language.

Returning to America in 1816 Dr. Walker married Eliza Hurd, daughter of Joseph Hurd of Charlestown. For fully thirty years Dr. Walker was a physician and surgeon in Charlestown. In his attractive home there on Main and Harvard Streets, with large grounds (near the original site of John Harvard's Charlestown home), Dr. Walker conducted a famous medical school. We have a long list of his students, many of whom became eminent physicians and surgeons. At Charlestown his eight children were born. The last survivor of his children, Miss Emily Hurd Walker, died at Pau, France, September 24, 1916. There one granddaughter, Catherine, Comtesse de Navailles, still lives. Another granddaughter, Mrs. E. W. Pudor, lives at Madison, Wisconsin.

Dr. Walker died April 2, 1865 at Newport, Rhode Island. He was buried in Mount Auburn in Lot 3637 on Mound Avenue. Yours truly,

139 Winthrop Road, Brookline, Mass.
HOSEA S. BALLOU.

*Figures refer to the different generations.

ARTICLES ACCEPTED BY THE AMERICAN MEDICAL ASSOCIATION COUNCIL ON PHARMACY AND CHEMISTRY

535 North Dearborn Street, Chicago, Ill.,
June 30, 1933.

Managing Editor,
The *New England Journal of Medicine,*

In addition to the articles enumerated in our letter of May 27 the following have been accepted:

Abbott Laboratories
- Sterile Ampoules of Procaine Hydrochloride Crystals (Abbott) 100 mg.
- Sterile Ampoules of Procaine Hydrochloride Crystals (Abbott) 120 mg.
- Sterile Ampoules of Procaine Hydrochloride Crystals (Abbott) 150 mg.
- Sterile Ampoules of Procaine Hydrochloride Crystals (Abbott) 200 mg.

Lederle Laboratories, Inc.
- Refined and Concentrated Antipneumococcic Serum Type II (Lederle)

Eli Lilly & Co.
- Extralin
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Yours sincerely,
PAUL NICHOLAS LEECH, *Secretary,*
Council on Pharmacy and Chemistry.

REPORTS AND NOTICES OF MEETINGS

AMERICAN PSYCHIATRIC ASSOCIATION

The eighty-ninth annual meeting of the American Psychiatric Association was held at the Hotel Statler in Boston on May 29, 30, 31, June 1 and 2, 1933. The President, Dr. James V. May, Commissioner, Department of Mental Diseases for Massachusetts, presided.

The section on convulsive disorders met on Monday, the twenty-ninth, and of the eleven papers read, six were by Massachusetts physicians.

The general meeting was called to order on Tuesday morning, May 30, 1933. Addresses of welcome were given by the Honorable James M. Curley, Mayor of the City of Boston, Dr. Halbert G. Stetson, President Massachusetts Medical Society, Dr. Morgan B. Hodskins, President, Massachusetts Psychiatric Society, and the Honorable Herbert C. Parsons, President, Massachusetts Mental Hygiene Society. The response was by Dr. James V. May, President of the American Psychiatric Society.

Following this there was time given for memorials to deceased members and after this the presidential address was given.

Dr. May said, in part: "The time has come when we must decide whether this Association is to assume the responsibility for the establishment of definite psychiatric standards which will unquestionably be generally recognized and accepted in this country.

or whether we are willing to leave the solution of these important problems to others."

Later in the meeting his recommendation was taken up for action and a board of prominent psychiatrists was appointed to arrange for the examination and the crediting of those whose training and experience were such that they might be considered competent to practice the specialty of psychiatry.

Dr. May also discussed the matter of expert testimony and commented upon the value of Massachusetts Laws in this respect.

The remainder of the day was spent in the presentation of studies in pathology and methods of treatment of mental diseases by psychotherapy.

On Wednesday morning a joint session was held with the American Psychoanalytic Association and the papers presented had to do with the psychoanalytic point of view.

In the afternoon the American Association for the Study of the Feeble-Minded presented its program and in the second session of this day the papers had to do with the interpretation of symptomatology and studies in Schizophrenia.

On Wednesday evening the annual dinner was held at the New Ocean House, Swampscott, Massachusetts. Governor Ely was present and welcomed the Association on behalf of the Commonwealth. Registration for this session was one of the largest in years.

On Thursday, papers on Child Guidance were given and clinical studies of mental tests. There were also practical papers given on administrative matters within the mental hospitals and the various forms of therapy, and in the afternoon papers on blood chemistry and studies of certain drugs and their reaction upon the nervous system. Other papers had to do with mental hygiene, mental nursing and studies in psychobiology.

Round table discussions were held on Thursday evening.

On Friday treatment of post-encephalitis, mental disturbances after head injury, and psychiatric study of prisoners were taken up.

Officers for the coming year were elected as follows:

President, Dr. George H. Kirby, Director of the New York State Psychiatric Institute and Professor of Psychiatry at Columbia University; Vice-President, Dr. C. F. Williams, Superintendent of the State Hospital at Columbia, South Carolina; Secretary, Dr. William C. Sandy, of Harrisburg, Pennsylvania, Director of the Bureau of Mental Health in the Pennsylvania Department of Welfare. Dr. Ransom A. Greene, Superintendent of the Walter E. Fernald State School of Waverley, was chosen President of the American Association for the Study of the Feeble-Minded.

The Committee on Arrangements was as follows:

Chairman, Dr. L. Vernon Briggs, Boston; Vice-Chairman, Dr. William A. Bryan, Worcester; Secretary, Dr. Winfred Overholser, Wellesley Hills; and Dr. C. Macfie Campbell, Boston; Dr. Ralph M. Chambers, Taunton; Dr. Charles H. Dolloff, Concord, N. H.; Dr. Albert Evans, Boston; Leopold M. Goulson, Esq., Boston; Dr. Donald Gregg, Wellesley; Mrs. Claire H. Gurney, Wollaston; Dr. Carl J. Hedin, Bangor, Maine; Mrs. George M. Kline, Boston; Dr. Roy L. Leak, Middletown, Conn.; Dr. George E. McPherson, Belchertown; Dr. Arthur Noyes, Howard, R. I.; Dr. C. Stanley Raymond, Wrentham; Dr. Arthur H. Ring, Arlington Heights; Dr. Horace G. Ripley, Brattleboro, Vermont; Dr. Harry C. Solomon, Boston; Dr. E. A. Stanley, Waterbury, Vermont; Dr. A. Warren Stearns, Boston; Mrs. Nathaniel Thayer, Boston; Dr. Douglas A. Thom, Boston; Dr. Forrest C. Tyson, Augusta, Maine; Dr. W. Franklin Wood, Waverley; Mrs. Eva M. Watson, Boston; Mrs. Alfred B. Williams, Taunton; Mrs. Edna Dreyfus, Brookline; and Mrs. Katherine Devine, Milton.

THE AMERICAN BOARD OF OTOLARYNGOLOGY

An examination was held in Milwaukee, Wisconsin, June 12, 1933, during the meeting of the American Medical Association. Forty-eight candidates were examined, of whom nine were conditioned or failed.

The Board will hold an examination in Boston, September 16, 1933, just prior to the meeting of the American Academy of Ophthalmology and Otolaryngology. Prospective applicants for certificate should address the Secretary, Dr. W. P. Wherry, 1500 Medical Arts Building, Omaha, Nebraska, for proper application blanks.

H. P. Mosher, M.D.,
President,
W. P. Wherry, M.D.,
Secretary-Treasurer.

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

July 24-31—The IVth International Congress of Radiology. See page 1071, issue of May 18.

August 10, 11, 12—American Delegation to the International Goiter Conference. See page 768, issue of April 6.

August 13-27—Courses at the Tomarkin Foundation. See page 1070, issue of May 18.

September 16—American Board of Otolaryngology. See notice above.

September 29 and 30—New England Surgical Society, at Boston.

DISTRICT MEDICAL SOCIETY

MIDDLESEX SOUTH DISTRICT MEDICAL SOCIETY

July 19—A joint meeting of the Middlesex South District Medical Society and the Middlesex South District Medical Golf Association will be held at the Weston Golf Club, Wellesley Street, Weston. Luncheon 12 noon. An address will be given by Dr. Tehyi Hsieh on "Manchuria—Inside Out." Following the address the Golf Association will hold a business meeting for the election of officers.

BOOK REVIEWS

Annals of Roentgenology edited by James T. Case. Volume Ten, Peptic Ulcer. Revised and published in February, 1933, by Jacob Buckstein, M.D. Second Edition. Paul B. Hoeber Publishing Company, New York, N. Y. 417 Pages. 404 Illustrations. Price $12.00.

As in his previous edition, the author has presented the subject by means of representative case histories and roentgenograms with operative and pathological findings, a method which illustrates the various points of roentgen diagnosis in an admirable manner.

Chapters one and two describe the work of outstanding investigators in gastric examination by means of the roentgen ray and the development of our present knowledge of the roentgen signs of ulcer. These signs he then proceeds to illustrate with well-selected roentgenograms, diagrams, case histories, operative findings and pathologic reports.

He discusses the subject of chronic gastritis and its association with ulcer describing the work of Forssell on the variations in the gastric rugae, their radiating and hyperplastic appearance as seen in peptic ulcer. He also describes and illustrates the roentgen signs of healing and the differential diagnosis between benign and carcinomatous ulcers. The author believes that benign ulcers occasionally occur on the greater curvature of the stomach.

Chapter three is devoted to duodenal ulcer. From a description of the normal bulbus duodeni, both anatomical and roentgenological, the author goes on to describe and illustrate the deformities produced by ulcer. He explains by drawings how the deformity is produced by spasm, and scar tissue contraction, and discusses the normal and hyperplastic changes in the duodenal mucosa. In this chapter, as in the previous chapter on gastric ulcer, the author is careful to warn the reader against conditions which might simulate ulcer.

Chapter four deals with gastrojejunal ulcer. The author states that although the niche and the haustrocolic or jejunocolic fistula are the only positive signs of ulcer in this region, there are secondary signs which, taken together, may enable one to make a diagnosis. These signs are gastric or jejunal retention after six hours, hyperperistalsis, dilatation or narrowing of the jejunum, localized tenderness and fixation. He then illustrates by a number of cases the healing of jejunal ulcer coincident with the disappearance of symptoms.

The author concludes with several case histories and roentgenograms illustrating gastrocolic, gastrojejunocolic fistulae, and the complications of duodenal ulcer. There is also an extensive bibliography.

This book is recommended to the roentgenologist, surgeon and clinician as an excellent summary of our knowledge in this field of diagnosis.

Abortion: Legal or Illegal? By A. J. Rongy, M.D., F.A.C.S. Published by The Vanguard Press, New York, New York. 1933. 212 Pages. Price $2.00.

As might be assumed from his title, Rongy advocates the legalizing of abortion for other reasons than the preservation of the health of the mother. He argues for the legalization of abortion in illegitimate pregnancy, in cases of rape and incest, in cases of mental deficiency of the mother, in cases of desertion by the father or of the father's death during his wife's pregnancy, and even in order to limit the size of families for economic reasons or to preserve the mother's health.

In preparing a basis for this rather radical position, Rongy reviews the history of abortion and describes conditions as they now exist. He accepts the estimate that the number of abortions in this country is around two million annually; of these only about five per cent are therapeutic abortions. Society, he says, has changed in its attitude toward the abortionist; "far from being looked down upon as a scoundrel, (he) is regarded as a sort of benign Robin Hood who defies the law to help the needy". The medical profession, according to Rongy, is considerably perplexed by the problem. Its members see abortionists getting rich quickly, and its younger members are often tempted by the financial reward which they could so easily secure. Ex-abortionists, because of their wealth, are able to secure for themselves influential positions on the staffs of hospitals. The average physician, moreover, approves at heart of broadening the limitations on this method of terminating pregnancy, but is prevented from saying so by his inherited inhibitions and his fear of alienating his clientele.

Contraceptive methods of controlling births are dismissed by Rongy as inadequate, unreliable and destructive to the emotional state which should prevail at such times. Sterilization should be done much more frequently.

We hardly believe that Rongy's book will be received with general approval. We feel that his interpretation of modern conditions is unduly favorable to his thesis, and that he fails to take into account those arguments which are against it. It is significant of the changing times, however, that a book teaching such doctrines as this has been published at all.

Medical State Board Examinations. By Harold Rypins, M.D. Published by J. B. Lippincott Co. Price $4.50.

As this is the first published work of its kind, "an organized review of actual questions given in medical licensing examinations throughout the United States," it deserves perhaps more than passing comment. Undoubtedly in a number of medical schools there were given in the past, such "or-

ganized reviews," printed sometimes but not published and the students took great comfort in the recapitulation in preparation for the crisis of the state board examination. They have been pretty generally given up as the proper functions of state examining boards and of medical schools and have become recognized more clearly as not identical. The book is not a compilation of questions and answers, but a topical and somewhat systematic treatment of subjects that examiners throughout the United States in the past ten years have thought should be considered and concerning which knowledge has been regarded as necessary for practice. It is a distinct improvement on the usual compilation. It gives, however, a somewhat distorted view of medicine: for example, there is no reference to the first cranial nerve. But the result of Dr. Rypins' efforts is a manual likely to prove very useful to some candidates for examination. The book does not lend itself easily to cramming, though it may be used for that purpose, but it would be helpful for a review of what the student has once known and has in part forgotten. For purposes of review it may receive a hearty welcome. The percentage of errors is perhaps not unduly high for the first edition of such a book and a second edition will see many of these corrected. The quite consistent use of "breach" for "breech" in obstetrics is a noteworthy slip.

The Colon, Rectum and Anus. By Fred W. Rankin, J. Arnold Bargen and Louis A. Buie. Published by W. B. Saunders Co. 1932. Price $9.50.

This book, coming as it did from the press with little or no advance publicity, bids fair to remain as a live textbook for many years because of its inherent worth. It represents the experience of the Mayo Clinic as expressed through the ideas of three men whose individual experience in their respective fields has been usually large, and who have been afforded opportunities for accurate observation and careful follow-up met with in few, if any, other clinics or hospitals. It is of course impossible nowadays to write an eight hundred page book on any of the well-known regions of the body without repeating a great deal that has been said before. This book contains, however, a wealth of new material and liberal reference to the labors of others.

The extensive work of Bargen on chronic ulcerative colitis, presented in numerous publications since 1927, is here coördinated in a single long chapter. His results in treatment are remarkably good. The use of warm saline colon irrigations is advocated in preference to any other solutions. A vaccine of Bargen's diplostreptococcus was used in most cases, combined with elimination of foci of infection, and with the institution of proper feeding. Ileostomy was performed in a limited number of cases, and is preferred above any other drainage operation.

Roughly one fourth of the book is devoted to a consideration of *benign and malignant tumors.* Polyposis is dealt with in a separate chapter, and the visualization of polyps by Fischer's method of x-ray is nicely illustrated. Carcinoma is thoroughly discussed.

The *operative procedures* for cancer and all the minor rectal conditions are dealt with in the last chapter which embraces one hundred pages.

The subject of *hemorrhoids* is presented in a very sane manner. The use of injection treatment primarily in cases of uncomplicated internal hemorrhoids is emphasized, and in the operative treatment no brief is held for a particular operation but rather for the proper performance of whichever one the operator likes and does best.

Good results with alcohol injection in *severe pruritus* cases are reported. This method of treatment is used, however, only when simpler methods of treatment have failed to give much relief of itching.

Ten operations of sympathetic ganglionectomy for *megacolon* have been carried out without mortality, but end results over a period of time are not available. This avenue of attack gives the greatest promise of relief for a most distressing condition.

The *granulomatous diseases* are thoroughly discussed in a chapter of fifty pages, and chapters are devoted to anal infections, diverticulosis and diverticulitis, parasitic diseases, functional disorders, volvulus, intussusception, methods of diagnosis, pre- and postoperative treatment, etc.

This book ranks with the best of its kind.

Practical Hematological Diagnosis. By O. H. Perry Pepper, M.D., Professor of Clinical Medicine, University of Pennsylvania; and David L. Farley, M.D., Physician to the Pennsylvania Hospital, Philadelphia. 562 Pages. Philadelphia and London: W. B. Saunders Company, 1933. Price $6.00.

The authors begin well in their introduction by emphasizing the important fact that the term "diseases of the blood" is outmoded, since it is not that the blood itself is diseased but that the tissues contributing to its condition are modified. This hint of a description of blood pictures based upon an underlying physiological pathology is unfortunately not borne out. There is no clear and detailed description of the blood-forming tissues, although descriptions of the blood cells are detailed and accurate. The chief features of the book are its systematic classifications and its great dependence upon a carefully-scanned literature. There is frequently too much verbose quotation and not enough of the authors' personal experience. The various hematological methods are well described. It is to be regretted that the authors transplanted piecemeal many of Gradwohl's very quaint "English" translations ("stab" form, "juvenile" form, etc.) of Schilling's fine work on the blood-picture. The sections dealing with the blood-platelets and the purpuras are unusually good. In regard to the anemias, there is too much involved classification

and too little dependence upon broad groupings based upon physiological principles. This holds true for the leukemias. The last part of the book (150 pages) deals with an alphabetical list of blood-pictures, complete, but of doubtful value. The very few illustrations are poorly drawn and exceedingly poorly lithographed. There is a good "additional hematological terminology" and an excellent bibliography and index.

As a work on "peripheral" hematology, the book is complete and contains the most recent developments in hematological practice. Hematology, however, has long ago advanced away from its narrow field as a study of the peripheral blood. Thus "Practical Hematological Diagnosis," although a new book, is fundamentally old, and although it will undoubtedly prove useful, is certainly not inspiring.

Endocrine Medicine. By WILLIAM ENGELBACH. Vol. 1, General Considerations; Vol. 2, The Infantile Endocrinopathies—The Juvenile Endocrinopathies; Vol. 3, The Adolescent Endocrinopathies — The Adult Endocrinopathies; Vol. 4, Bibliography. Index. Published by Charles C. Thomas. Price $35.00.

For several years there has been no good, up-to-date, concise textbook of endocrinology in the English language. Our German colleagues have been much more fortunately situated and have had several excellent works to which they could refer (e.g., Zondek, 1927; Bauer, 1927; Falta, 1928). Dr. Barker in the preface to the late Dr. Engelbach's work stated, "It requires bravery, and prodigious, concentrated industry, . . . to attempt to master, and to summarize for others, the present status of the anatomy and physiology of the endocrine glands; that of the clinical syndromes that have been described and of the pathological-histological changes and the alterations in body chemistry and physics that underlie them; and that of the relations of the internal secretions to heredity, to constitution, and to the biology of the person and of the race."

Dr. Engelbach has attempted all this and more; has perhaps given his life in this tremendous undertaking; and yet, in the opinion of the reviewer, has in many ways failed. This author not only covers the entire field, but does so for three different age periods (infants, adolescents, and adults). This leads to endless repetition. One finds citations of well-proved autopsied cases followed by other purely clinical cases, where the diagnosis seems most uncertain. The terminology used is perhaps indication of the work. One finds, for example, "hypergonadism," which conveys a definite thought, and in another page "thyroidism," which is meaningless. In spite of these criticisms, the reviewer has found the work of considerable value as a reference book.

La Gymnastique des tout Petits. By DORIS REICHMANN. Translated from the German by Dr. P. Gauthier-Villars and published by Gauthier-Villars & Cie. Price 20 fr.

This little book of 57 pages describes, chiefly by means of excellent illustrations, a series of exercises for infants. The need of such exercises is based on the supposition of the fetus in utero, stretches the dorsal and extensor muscles of body and extremities and allows the ventral and flexor muscles to relax and atrophy. Consequently exercises both passive and resistive in type are needed to develop a normal balance between the two groups of muscles. Whether the position in utero seriously disturbs such a balance, times arise when because of malnutrition or convalescence from disease such exercises would be of value. The value of exercises during convalescence is well recognized in the case of adults but is seldom considered in infants because of the difficulty of getting the patients' coöperation. Much of this difficulty should be eliminated by a study of this little book. The arrangement of the book is very simple and clear. Each exercise is illustrated by two or more photographs and there is a paragraph on how to execute the maneuver, the precautions to be observed, and the purpose of the exercise. The author believes that all the exercises described can be safely carried out by the mother. This seems to assume a higher maternal intelligence than is sometimes encountered.

A book of this sort will never have the vogue that similar ones written for children and adults have had, but it should be a great help to the pediatrician, the orthopedist, and perhaps the neurologist who may occasionally wish to employ exercises in treating the very young.

Gastric Anacidity. Its Relation to Disease. By ARTHUR L. BLOOMFIELD, M.D., and W. SCOTT POLLARD, M.D. Published by the Macmillan Co. Price $2.50.

It is refreshing to read a book such as this in which an honest attempt has been made by well-qualified authors to simplify and elucidate a subject long befogged by tradition and by incomparable superficial observations. The subject has been stripped of much that was futile and imperfectly controlled. True anacidity has been clearly and simply defined, and the condition discussed in relation to gastric physiology, and certain diseased states, notably pernicious anemia, gastric cancer and otherwise unexplained associated diarrhea. The wide prevalence of true anacidity and reduced acidity among otherwise normal subjects has been carefully stressed, particularly in relation to an underlying "gastritis". An explanation of the gradual increase in true anacidity with advancing years should be based upon the existence of "an involutional phenomenon of no special clinical significance," according to the authors who have successfully swept away many of the existing sup-

positions as to the clinical seriousness of such a disturbance in gastric secretion. While admitting that anacidity is a possible precursor and cause of pernicious anemia and cancer, they rightly point out that such a conception is still not substantiated by convincing proof. Treatment of anacidity as such unless associated with diarrhea, they rightly conclude, requires no special treatment. The book is written in a critical but simple fashion and represents a distinct and convincing addition to the literature. The bibliographies have been carefully chosen in a thorough and discriminating manner.

Les Ictères. By ÉTIENNE CHABROL. Masson et Cie, Editeurs, Paris, 1933. Price 75 fr.

This is a splendid volume covering all phases of jaundice. Professor Chabrol, who is well known for his work on hepatic pathology, has succeeded in writing a book that should be interesting to clinicians, surgeons, and general practitioners alike. Laboratory technicians will find this book exceedingly helpful for a section in the first chapter deals with the chemistry of bile salts, bile pigments, and liver function tests. Careful and critical analyses are made of these tests and their practicability evaluated. The author states that a moderate amount of carotin and lutein in the blood serum may sometimes give the clinical picture of jaundice and cautions against making this error.

The last chapter is entirely devoted to therapy. Duodenal drainage, intestinal antiseptics, and cholagogues are just a few of the medical treatments discussed. The section devoted to surgical treatment is largely borrowed from the experiences of competent abdominal surgeons such as Professor Hartmann. The latter states that the mortality incident to cholecystectomy and choledochostomy is no greater than with simple choledochostomy. He uses chloroform in preference to ether as the anesthetic of choice.

The reviewer feels that the book is exceedingly well done, and an inestimable aid for reference.

Surgery of the Chest. By GEORGE F. STRAUB. Published by Charles C. Thomas, 1932. Price $10.50.

This work is more ambitious in its scope than the sections devoted to Thoracic Surgery included in the larger systems of surgery, although it falls far short of being an authoritative monograph of interest to special students of the subject.

The author purposely refrains from a comprehensive exposition of anatomic, physiologic or pathologic facts pertinent to the subject in an effort to make his book of practical value. Even if this serious shortcoming be overlooked, the text cannot be considered a sound one for a surgeon desiring to qualify himself to undertake the operative treatment of thoracic disease, largely because it fails to give an adequate perspective of the dangers and difficulties that attend many of the major operative procedures that are presented. Operations that have been successful only in a very few fortunate cases at the hands of very experienced operators are described and illustrated in a convincing manner. The impression is gained that the subject matter of the book has been culled from a very complete survey of the literature rather than from a seasoned personal contact with the hazards and difficulties that attend clinical application of theoretical procedures. The publishers are to be commended for the excellence of their efforts. The illustrations, many in color, are graphically drawn and clearly reproduced.

New and Nonofficial Remedies. 1933, containing descriptions of articles which stand accepted by the Council on Pharmacy and Chemistry of the American Medical Association on January 1, 1933. Cloth. Price, Postpaid, $1.50. Pp. 498; lvi. Chicago: American Medical Association.

This publication represents a valuable function of the American Medical Association exercised by its Council on Pharmacy and Chemistry, in that it places before the profession the results of its study of the various articles submitted for approval based both on the quality of the product and the text of the arguments designed to promote sales.

The book presents much information respecting approved products and shows clearly the reasons for rejecting those which are not of value.

The conclusions of the Council are the combined opinions of men with scientific training and should be helpful to practitioners who are desirous of using ethical preparations.

The Law Against Abortion. By WILLIAM J. ROBINSON. The Eugenics Publishing Co., Inc. New York. Price $2.00.

"It is time that the justifiability of abortion . . . be discussed in a calm, judicial, scientific and above all humane spirit." On this point there can be little difference of opinion among calm, judicial, scientific and humane persons, although they might not agree on what to do about it after the discussion. This book by Dr. Robinson is a plea for and a demonstration of the need of such discussion.

"Now the last word has been spoken on the subject" of abortion (p. 30). Infallibilism is provocative and sometimes provoking. Dr. Robinson's brand of infallibility is not likely to receive widespread acceptance. "An inanimate pinhead of cells which may potentially become a man" (p. 34). It is because this "pinhead of cells" is so generally regarded as animate that many persons object to abortion. "The truth is generally a safe guide"

(p. 76). The irony here is misplaced. In Dr. Robinson's casuistics, he ignores any personal responsibility on the part of the mother. It is the "criminal law" alone which brings about the unhappy ending. Without apparently any knowledge of the actual situation in Russia, Dr. Robinson commends the Russian procedure in regard to the legitimizing of abortion.

The book contributes little to the scientific discussion of abortion, and seems not likely to stimulate the kind of discussion that is needed.

Diseases of the Heart. The Methods for Their Diagnosis and Treatment. By WILLIAM D. REID, M.D., F.A.C.P., The Graphic Press, Newton, Mass. 1933. Pp. 105 including index, paper covers. Price 65 cents.

The purpose of this booklet is, as stated by the author, to supply the need for a guide or laboratory manual for students and general practitioners rather than a textbook on heart disease. There are nine chapters and an appendix, and what is considered of special attention is accented by vertical lines in the margins of the text. The material serves as an introduction for students to the general methods employed in the study of patients with heart disease and progresses at intervals by the use of the rhetorical question and answer. Much of the language is colloquial, and some of it very fauity in construction, but it makes for easy reading and ready emphasis. Issue might be taken with some of the observations such as those of the x-ray findings in certain types of heart disease, and the description of "senescent" heart disease which gives a very incomplete picture of coronary arteriosclerosis of middle age. The text is marred for the careful reader by a score or more of errors in spelling and proof reading. However, the student or practitioner with a good knowledge of the material of this booklet would have a sound point of view in approaching cardiac problems.

The Nervous Child at School. By HECTOR CHARLES CAMERON, M.A., M.D., F.R.C.P. Oxford University Press, 1933. Price $1.50.

Dr. Cameron has been interested in the "Nervous Child" for many years and his conclusions are based on ripe experience. To those who are somewhat appalled at the elaborateness of some of the current methods of child guidance, the volume under review is cheerful. Dr. Cameron thinks that life is comparatively simple, he is dogmatic about matters which often seem complicated and he is jocosely intolerant of the highly organized clinic.

He stresses the importance of fatigue and acidosis. In spite of the author's experience, the reviewer left this volume with the feeling that it was a well done bit of special pleading for a limited point of view and does not believe that the argument justifies the conclusion that the management of fatigue is of paramount importance in bringing up children. Taken as a whole this sequel is far less useful than its predecessor: "The Nervous Child."

Nouveau Traité de Médecine. Published under the direction of G. H. Roger, Fernand Widal and P. J. Teissier. Fascicule X. (Pathologie de l'Appareil Circulatoire.) Tome III, Vaisseaux. Masson et Cie, Editeurs. Price 115 fr.

This volume consisting of 700 pages gives considerable space to Aortitis. There is an excellent chapter on "Aneurysm of the Aorta." Tension Arterielle is considered from all angles and composes approximately one-fourth of the book. There is a very excellent chapter on Arteritis with more said about syphilitic arteritis than has hitherto been published. The section on Thrombo-Angiitis Obliterans is short and adds nothing new. Strangely enough only very slight mention is made of Raynaud's Syndrome.

There is a good section on diseases of the venous system including phlebitis and treatment of varicose conditions.

This work is apparently well done and quite authentic, stressing the foreign authorities on various subjects. It will be a volume valuable for reference to one interested in the diseases of the large blood vessels.

Binocular Vision and the Modern Treatment of Squint. By MARGARET DOBSON, M.D. Published by the Oxford University Press. Price $2.75.

Strabismus is one of the problems in which an increase in the knowledge of causes will aid in the intelligent treatment of the condition. Dr. Dobson's treatise summarizes the present status of knowledge about strabismus, and deals with the non-surgical treatment to be applied to strabismic patients. The delayed attention of a qualified ophthalmologist in these cases is properly deplored. Attention is given to the modus operandi of suspension of vision and of amblyopia.

The value of orthoptic training is emphasized, for both the pre-surgical and the post-surgical periods in strabismus, and a chapter is devoted to the instruments and the methods in orthoptic training employed by the author.

While some workers may not be willing to accept the technic used by the author, the treatise is at least provocative of thought, and should thereby be useful.

BOOKS RECEIVED FOR REVIEW

An Elementary Handbook on Radium and Its Clinical Use by D. F. Clephan and H. M. Hill. Published by Oxford University Press. 164 Pages. Price $2.00.

The Surgical Clinics of North America. April, 1933. Volume 13, Number 2. New York Number. Published by W. B. Saunders Company. 274 Pages.

Diseases of Tradesmen by Bernardino Ramazzini. Compiled by Herman Goodman. *Silk Handlers' Disease of the Skin* by Herman Goodman. Published by the Medical Lay Press. 95 Pages.

The Physiological Effects of Radiant Energy by Henry Laurens. Published by The Chemical Catalog Company, Inc. 610 Pages. Price $6.00.

The Law Against Abortion by William J. Robinson. Published by The Eugenics Publishing Company, Inc. 123 Pages. Price $2.00.

The International Medical Annual. Edited by Carey F. Coombs and A. Rendle Short. 1933. Fifty-First Year. Published by William Wood and Company. 572 Pages. Price $6.00.

Gastric Anacidity. Its Relations to Disease by Arthur L. Bloomfield and W. Scott Polland. Published by The Macmillan Company. 188 Pages. Price $2.50.

The Vitamins in Health and Disease by Barnett Sure. Published by The Williams & Wilkins Company. 206 Pages. Price $2.00.

New and Nonofficial Remedies, 1933. Published by The American Medical Association. 498 Pages. Price $1.50.

Annual Reprint of the Reports of the Council on Pharmacy and Chemistry of the American Medical Association for 1932. Published by the Press of the American Medical Association. 104 Pages. Price $1.00.

Obstetrics and Gynecology. Edited by Arthur Hale Curtis. Volume I. Published by W. B. Saunders Company. 1165 Pages.

Surgical Pathology by William Boyd. Third Edition. Published by W. B. Saunders Company. 866 Pages. Price $10.00.

Urine and Urinalysis by Louis Gershenfeld. Published by Lea & Febiger. 272 Pages. Price $2.75.

History of Urology. Prepared under the auspices of the American Urological Association. Volumes I and II. Published by The Williams & Wilkins Company. 385 Pages in Volume I and 361 Pages in Volume II. Price $8.00 for both volumes.

Diet in Sinus Infections and Colds by Egon V. Ullmann. Published by The Macmillan Company. 166 Pages. Price $2.00.

Traité de Physiologie Normale et Pathologique. Published under the direction of G.-H. Roger and Léon Binet. Tome IX, *Système Nerveux.* Published by Masson et Cie. 566 Pages. Price 80 and 100 fr.

Internal Derangements of the Knee-Joint by A. G. Timbrell Fisher. Second Edition. Published by The Macmillan Company. 205 Pages. Price $3.50.

Arteriosclerosis. A Publication of The Josiah Macy, Jr., Foundation. Edited by Edmund V. Cowdry. Published by The Macmillan Company. 617 Pages. Price $5.00.

Outline of the Cranial Nerves by John Favill. Published by The University of Chicago Press. 112 Pages. Price $1.50.

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